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EPIDERMOID CARCINOMA OF THE PHARYNX, BUCCAL MUCOSA AND LARYNX*

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This is a brief statistical report of 169 cases of epidermoid carcinoma, involving the buccal mucosa, pharynx and larynx. The patients cited have been observed for periods of three to seven years in the Department of Otolaryngology and Oral Surgery at the State University of Iowa. The irradiation was carried out by the Department of Radiology under the direction of Dr. H. Dabney Kerr.

Except in a few instances, the cases were inoperable because of the extensiveness of the primary lesion, metastases, or the poor general condition of the patient. Consequently, for the most part, irradiation was the only treatment possible. In some cases, this treatment was augmented by surgery or electrocoagulation.

This paper will consider a few observations pertaining to one particular group of cases. Some reports and statistics of others also have been included.

Many, including Coutard[1] and Quick,[2] believe that early cancer is as favorable for irradiation as for surgical treatment. It is our opinion that for the present, best results in localized and differentiated tumors without metastases may be obtained by surgical pro-

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cedures, with or without irradiation. In this connection, it is only fair to say that better results with irradiation are achieved each year, so that this statement may not hold true in the future.

At the present time, many months may elapse between the onset of the disease and the first treatment, so that the real problem is the more advanced case, in which we have never been interested as far as surgery is concerned.

Many factors determine treatment and prognosis. McCarty[11] has summarized these very well. They include "presence or absence of lymph node involvement, fixation of growth, anatomical location, renal efficiency, presence or absence of anemia, cardiac efficiency, size, age, duration and direction of the growth, loss of weight, lymphocytic infiltration, fibrosis, hyalinization and cellular differentiation."

In this particular group, we were impressed by the fact that the average time which elapsed from the onset of the disease to the first admission to the hospital was between seven and eight months. Doubtless the economic status was a factor in the postponement of treatment, since most of these patients were from the indigent service.

The lesions in most cases present quite definite symptoms and usually can be seen by the simplest examination. But early indications usually had been disregarded, and not until they became concerned over later symptoms of pain and discomfort, did patients seek advice.

The youngest patient was 35 years old, and the oldest, 85. There were metastases in 91 cases, further indicating the extensive involvement in this series of cases.

Nothing new was learned regarding etiology and symptoms. As might be expected, poor hygiene, chronic irritation and infection were indirect factors.

In general, the primary lesions in undifferentiated tumors responded well to treatment, but in most cases, metastatic glands appeared which were not evident, but probably were present at the time the primary growth was treated. In general, better results were obtained in older persons, and in well-differentiated tumors. Less differentiated lesions, as would be expected, were seen oftener in younger patients.

As a rule, the tumors could be graded according to differentiation, but occasionally, varying degrees of differentiation could be found in the same block of tissue. Certain tumors of the same type or degree of differentiation appeared to vary in reaction to the same

degree of irradiation. We have not seen any evidence to support the contention that irradiation produces metastatic deposits.

Our results in general were poor and discouraging so far as so-called cures were concerned. It must be understood, however, that they were for the most part advanced, hopeless cases with no treatment possible except irradiation. It must also be pointed out that despite instances of discomfort and severe reaction from irradiation, life was prolonged many months, with, at least, an abeyance of symptoms.

The following tables contain statistics indicating results in the treatment of carcinoma of the larynx.

TABLE 1

LARYNX TREATED BY IRRADIATION

	Living	Dead	Total
1931-32-33	0	15	15
1934	1	5	6
1935	1	4	5
	2	24	26

This table reveals that all cases treated by irradiation in 1931-32-33 are dead. Two, or 7.6 per cent of the entire number treated in 1934-35, are living after three and four years.

Most observers find, at least for the present, that differentiated intrinsic cases respond better to surgery than to irradiation. Early differentiated cases may be treated without loss of function.

Ten selected cases were treated surgically in 1931-33, and all except one are alive today. This percentage is high, and over a period of years will be considerably lower. However, MacKenty,[4] in 1927, in 102 laryngectomies, reported 79.4 per cent free from recurrence after five years. Thomson,[5] in 1930, reported 76 per cent three-year cures by laryngofissure. Jackson[6] and others have had excellent results with surgery.

Table 2, which follows, indicates clearly the delay between first symptoms, and application for treatment.

Fourteen, or 35 per cent, of the laryngeal cases were operable. There were metastases in 15 cases. It is rather appalling that seven months elapsed between the onset of the disease and the time of treatment, in the average case. Many patients, if seen earlier, could have been spared radical treatment, with preservation, to a large degree, of laryngeal function.

TABLE 2

Average time from onset to first admission	7 months
Shortest time	3 months
Longest time	12 months
Average age	60 years
Youngest	42 years
Oldest	72 years
Metastases	15 cases

Treatment	Living	Dead	Living	Dead	Living	Dead
Surgery	7	1	0	0	2	0
Surgery and Irradiation	4	0	0	0	0	0
Irradiation	0	15	1	5	1	4
	11	16	1	5	3	4

Kramer[7] reported 59 cases of carcinoma of the larynx in which 58 per cent were free from involvement after three years. All types of treatment were used. It is his opinion that differentiated squamous cell carcinoma responds more favorably to surgery than to roentgen therapy. He does say that in early cases, with freely moving vocal cords, equally good results may be obtained by irradiation and surgery.

Thyrotomy, however, is the treatment of choice, because of economy, comfort of the patient, and the shorter convalescence involved.

Many recommend a combination of irradiation and surgery in the treatment of these tumors. Kramer[7] has expressed the belief that irradiation should be given in cases of doubt following surgery as soon as the wound has healed. Hauntaunt[8] reported 322 cases, 200 of which were treated surgically, and 122 by irradiation. It is his contention that combination surgery and irradiation give the best results. He suggests preoperative irradiation to "sterilize the outlying parts of the tumor rather than to destroy it." He believes this delays healing, but has noted no serious complications.

Garfin[9] reviewed the results of 202 consecutive cases treated at the Collis P. Huntington Memorial Hospital in Boston, between 1919 and 1933. Total laryngectomy was done in seven patients, three of whom survived from three to four years. Laryngofissure was done in twelve cases, with one operative death. Four survived five to thirteen years. Twenty cases were treated with combined surgery and irradiation. Five of these were free of the disease after three years. Six months was the average length of survival for 33 extrinsic cases, treated with radium. Roentgen therapy was used in 37 advanced cases, and only four were living after one year. Combination radium and roentgen therapy was used in the treatment of

40 other cases, 12 of whom lived longer than one year, but only one of whom survived five years.

Coutard[1] reported 39 out of 142, or 27 per cent, five-year cures of carcinoma of the larynx. His poorest year was 1922, when none was living after five years. In 1932, six out of nine, or 66 per cent, were living after a five-year period. His ten-year record, in 77 selected cases, was 23.16 per cent living. New and Waugh[10] had 82.3 per cent five-year cures in which thryotomy had been done, and 56.1 per cent cured by laryngectomy.

TABLE 3

PHARYNX

	Living	Dead	Total
1931-32-33	1	10	11
1934	0	1	1
1935	0	5	5
	1	16	17

Average time from onset to first admission	8 months
Shortest time	4 months
Longest time	12 months
Average age	57 years
Youngest	42 years
Oldest	73 years
Metastases	14 cases

	1931-32-33		1934		1935	
Treatment	Living	Dead	Living	Dead	Living	Dead
Surgery	0	0	0	0	0	0
Surgery and Irradiation	0	0	0	0	0	0
Irradiation	1	10	0	1	0	5
	1	10	0	1	0	5

Available statistics, including this small group, indicate that surgery, or combination surgery and irradiation, are the treatments of choice for intrinsic lesions, particularly those which are well localized and differentiated (Grade I and II). Occasionally, surgery also is done in selected extrinsic cases; but as a rule irradiation is the treatment of choice for this type of lesion.

Our next table, recording results in treatment of carcinoma of the pharynx, shows that only one of these cases, or 5.8 per cent, was living after seven years. All of these lesions were very extensive and were inoperable. There were metastases in 14, or 82 per cent, of the cases.

Furstenberg[11] reports forty cases of malignant neoplasms of the nasopharynx, only one of which survived after five years. He used

all types of treatment, including radical incision, electrocoagulation and irradiation. On the other hand, Cade and Allchin[12] reported 52 cases of malignancies of the pharynx treated by combination irradiation and radium bomb, 17 per cent of which were well after three and one-half years.

Coutard[1] suggested that tumors of the pharynx are more or less differentiated but of mucous membrane type and always accompanied by adenopathy. This may explain the difference in results found in contrast to laryngeal lesions. He also states that variations in the results in carcinoma of the larynx are six times as numerous as in carcinoma of the pharynx because the "diversity of types" is much greater in the larynx. Coutard's statistics in epidermoid carcinoma of the hypopharynx reveal 10 to 12 per cent five-year cures.

In most instances, any type of treatment has been unsatisfactory. However, best results seem to have been obtained by irradiation combined with surgery.

It will be noted in Table 4, that of eighteen cases treated for epidermoid carcinoma of the tonsil, one was alive seven years after being treated by surgery and irradiation, and one was alive three years after treatment by irradiation. Eleven per cent were still alive after three and seven years. Fifty-five per cent had metastases.

TABLE 4

EPIDERMOID CARCINOMA OF THE TONSIL

	Living	Dead	Total
1931-32-33	1	10	11
1934	0	3	3
1935	1	3	4
	2	16	18

Average time from onset to first admission	6 months
Shortest time	2 months
Longest time	12 months
Average Age	66 years
Youngest	51 years
Oldest	85 years
Metastases	10 cases

	1931-32-33		1934		1935	
Treatment	Living	Dead	Living	Dead	Living	Dead
Surgery	0	0	0	0	0	0
Surgery and Irradiation	1	1	0	0	0	0
Irradiation	0	9	0	3	1	3
	1	10	0	3	1	3

Norman Patterson[13] has reported 51 cases of carcinoma of the tonsil and its area. The treatment was diathermy for the primary growth and gland excision when necessary. Twelve persons, or 22 per cent, were alive after five years. Most observers reported the best results with diathermy and surgery in combination with irradiation.

Reports are uniformly more favorable for epidermoid carcinoma of the alveolar process, as the following table will indicate.

TABLE 5

EPIDERMOID CARCINOMA OF THE ALVEOLAR PROCESS

	Living	Dead	No Report	Total
1931-32-33	3	3	0	6
1934	1	2	0	3
1935	1	5	1	7
	5	10	1	16

Average time from onset to first admission	5½ months
Shortest time	1 month
Longest time	12 months
Average age	65 years
Youngest	42 years
Oldest	77 years
Metastases	8 cases

	1931-32-33		1934		1935	
Treatment	Living	Dead	Living	Dead	Living	Dead
None	0	0	0	0	0	0
Surgery	0	0	0	0	0	0
Surgery and Irradiation	2	1	0	0	0	0
Irradiation	1	2	1	2	1	5
	3	3	1	2	1	5

Three in this group were alive after five to seven years. Two of these were treated by surgery and irradiation. Two were treated by irradiation and were living after three and four years. There were metastases in eight, or 30 per cent, of the cases. Five out of sixteen, or 31 per cent, were alive from three to seven years following treatment.

Combination surgery and irradiation seemed best in cases of carcinoma of the hard and soft palate. After five to seven years, two patients who received this treatment were still alive. There were metastases in four, or 44 per cent, of the cases. The statistics on this group of cases follow:

TABLE 6

EPIDERMOID CARCINOMA OF THE PALATE (HARD AND SOFT)

	Living	Dead	Total
1931-32-33	2	4	6
1934	0	2	2
1935	0	1	1
	2	7	9

Average time from onset to first admission	13 months
Shortest time	10 months
Longest time	16 months
Average age	65 years
Youngest	51 years
Oldest	74 years
Metastases	4 cases

	1931-32-33		1934		1935	
Treatment	Living	Dead	Living	Dead	Living	Dead
Surgery	0	0	0	0	0	0
Surgery and Irradiation	2	1	0	0	0	0
Irradiation	0	3	0	2	0	1
	2	4	0	2	0	1

Difficulty in the curing of carcinoma of the buccal mucosa is indicated by the following figures:

TABLE 7

EPIDERMOID CARCINOMA OF THE CHEEK

	Living	Dead	No Report	Total
1931-32-33	6	28	0	34
1934	2	4	1	7
1935	0	4	2	6
	8	36	3	47

Average time from onset to first admission	5½ months
Shortest time	1 month
Longest time	12 months
Average age	62 years
Youngest	35 years
Oldest	78 years
Metastases	21 cases

Treatment	Living	Dead	Living	Dead	Living	Dead
Surgery	0	0	0	0	0	0
Surgery and Irradiation	4	2	1	0	0	0
Irradiation	2	26	1	4	0	4
	6	28	2	4	0	4

Eight of these patients, or 17 per cent, were living after four years. Of these, seven were treated by combination surgery and irradiation. Five are living after four to seven years. There were metastases in twenty-one cases, or 44 per cent.

Lund[14] reported sixteen cases with 38 per cent living after five years. He concluded that epidermoid carcinoma of the buccal mucosa is difficult to cure in most cases. He added that small, low-grade tumors are "susceptible to cure" by proper application of any recognized method. Out of 519 cases with small glands he reported 11 per cent cures.

Radiological methods have been used with some success in treatment of epidermoid carcinoma of the tongue. Statistics relative to our experience with a group of cases follow in Table 8.

TABLE 8

EPIDERMOID CARCINOMA OF THE TONGUE

	Living	Dead	No Report	Total
1931-32-33	0	12	0	12
1934	1	3	1	5
1935	3	2	1	6
	4	17	2	23

Average time from onset to first admission	6 months
Shortest time	1 month
Longest time	24 months
Average age	62 years
Oldest	77 years
Youngest	38 years
Metastases	19 cases

	1931-32-33		1934		1935	
Treatment	Living	Dead	Living	Dead	Living	Dead
Surgery	0	0	0	0	0	0
Surgery and Irradiation	0	0	0	0	0	0
Irradiation	0	12	1	3	3	2
	0	12	1	3	3	2

In this particular group the irradiation is the combination of radium needles and x-ray in every case which survived. The four cases listed as living are the four in which there were no metastatic lesions. Thus four cases out of twenty-three, or 17.3 per cent, were living after three to four years. These lesions were fairly well localized and differentiated.

Wookey,[15] reporting on epidermoid carcinoma of the tongue, mentioned control of the primary lesion by radiological methods, but admitted that sooner or later the regional glands became involved.

Lund[14] reported a series of forty-six cases of carcinoma of the tongue with 26 per cent cures. Norman Patterson[13] reported thirty-four cases of involvement of the tongue and the floor of the mouth, 26 per cent of which survived five years.

Gordon Richards,[13] in a discussion of Patterson's paper, reported 66 per cent cures in carcinoma of the base of the tongue; 24 per cent with involvement of the lateral margin; 33 per cent of the tip, and in the involvement of the dorsum have survived. These results were obtained by radiological methods only.

Gordon New,[13] discussing the same paper, felt that best results are obtained by irradiation, but that cautery, excision and diathermy offer good chances of recovery in low-grade lesions.

COMMENT

1. Well-localized, differentiated lesions for the present, at least, respond better to surgery, or surgery combined with irradiation.

2. Advanced malignancies and undifferentiated tumors do not respond well to any treatment. Irradiation, the only treatment possible, is definitely of value. Months or years of life with freedom from symptoms may be obtained.

3. Education in regard to early recognition of malignancies is one of the most important problems to be solved.

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LXXI

SEVERE INJURY TO THE LARYNX RESULTING FROM THE INDWELLING DUODENAL TUBE (CASE REPORTS)* **

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In his interesting history on "The Invention of Stomach and Duodenal Tubes," Paine[1] states that John Hunter, in 1790, was probably the first physician to make use of a stomach tube. For this purpose Hunter employed a fresh eelskin drawn over a whalebone probang. In Hunter's own words, "The probang thus covered was introduced into the stomach, and food and medicines were put into a bladder and squeezed down through the eelskin.[2] From this crude but ingenious device, the stomach tube took its origin and gradually attained its modern design and perfection after the invention of rubber.

The stomach tube had been in use for over a century when, in 1909, Gross[3] and Einhorn,[4] working independently, presented the duodenal tube to the medical profession. The duodenal tube differs from the stomach tube chiefly in its greater length and smaller diameter. The improved Levin tube (1921),[5] now in common use, is made of one piece of soft rubber and can be readily introduced through the nasal passage. The duodenal tube was first employed chiefly for diagnostic purposes in the study of gastroduodenal disease. As a later development, its use became general in the treatment of both medical and surgical abdominal conditions. At the present time the indwelling tube is in common use, either as a conveyor of food or drugs in patients unable or unwilling to swallow, or postoperatively as a siphon in the prophylaxis and treatment of gastric and intestinal

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distention. In 1931, Wangensteen[6] further improved the technique by adding the principle of continuous suction drainage. Recently, Miller and Abbott[7] have perfected a double lumen tube for intubation of the small bowel.

The postoperative use of the duodenal tube for the prophylaxis and treatment of distention has proved to be a great boon to both the patient and the surgeon. To quote Eusterman and Balfour,[8] "The progress of surgery in the last quarter century, however, is well illustrated by the fact that today death from dilatation (stomach) alone is one of the rarest complications of surgery." Patients usually tolerate the duodenal tube very well, and it is generally stated that the indwelling duodenal tube may be employed with impunity with no damaging by-effects. In their monograph, Eusterman and Balfour[8] reported that they had never observed any injurious effects on the larynx from the wearing of the tube. In his book on "Bowel Obstructions," published in 1937, Wangensteen[9] stated that "no harmful effects such as pressure erosion" had occurred in his practice. He stated, however, that infants might develop otitis media from the continuous presence of the tube in the pharynx.

Rankin[10] also stated that he had used the tube successfully without throat complications in hundreds of cases with but one exception (Case No. 2, this paper). Morawitz and Henning[11] reported that some patients complained of pharyngeal irritation, which disappeared when a thinner tube was substituted.

In the literature we have found but two references to laryngeal injury from the wearing of a duodenal tube. Morrison[12] in his recent textbook makes a brief statement to the effect that "cricoid chondritis may occur when a rubber feeding tube has been retained in the esophagus for long periods." The second reference is in the May, 1939, issue of Surgery, Gynecology and Obstetrics,[13] in which Wangensteen and his co-workers mention two cases which they have encountered.

While it undoubtedly is true that in the vast majority of instances the indwelling tube causes no damage and can be retained for prolonged periods, nevertheless we have found that a severe injury to the larynx is occasionally excited by the presence of the tube and that this complication may occur after a comparatively brief sojourn of the tube. During a period of two years we have, between us, observed ten cases of this kind. Eight of these cases developed laryngeal stenosis of such severity that tracheotomy became imperative. In this paper we present brief histories of these cases with pathological reports on two cases which came to autopsy.

REPORT OF CASES

CASE 1.—N. M. (No. 5129), aged 37, was admitted to St. Vincent's Hospital, Indianapolis, Nov. 19, 1936. A laparotomy was done Nov. 20, 1936, for fibroid tumor and chronic appendicitis. On Nov. 21, 1936, a Levin tube No. 12 was anchored for suction and was removed Nov. 27, 1936. The patient was re-operated for intestinal obstruction Dec. 18, 1936, at which time the tube was again anchored for suction only and was removed Dec. 25, 1936. The tube was re-anchored for twelve hours. Two days later the patient complained of pain in the right ear, sore throat, blood-tinged sputum, with hoarseness, odynophagia, all of which increased gradually in severity for one month, when it became necessary to perform a tracheotomy for relief of dyspnea.

A laryngeal examination just prior to the tracheotomy showed a generalized inflammation of the larynx with marked edema over the arytenoids and epiglottis. There was also tenderness over the thyroid cartilage. Wassermann was negative.

Present Condition: Examination by one of us (M) Nov. 1, 1938, found the patient still wearing a tracheal cannula. The vocal cords were fixed near the mid-line position and only a slight movement of the arytenoids was noted. Below the cords the larynx was completely occluded with fibrous or hypertrophic tissues. The patient was advised to have treatment for the relief of the stenosis, but has not yet consented.

Comment: This patient had two laparotomies performed in the course of thirty days. Nine days following the last operation and two days following the permanent removal of the Levin suction tube, involvement of the larynx became manifest, eventually requiring tracheotomy. One year later complete stenosis had developed. Surgical measures were advised for relief of the stenosis, but up to date nothing has been done. Friction of the duodenal tube, aggravated by debility, undoubtedly initiated the perichondritis followed by stenosis of the larynx.

CASE 2.—Mrs. M. S., a 62-year-old white woman, was operated upon Feb. 6, 1937, for a ruptured duodenal ulcer complicated with peritonitis. Shortly after operation, a No. 16 F. Jutte feeding tube was introduced into the stomach, and remained in place for eight days. Seventeen days after the laparotomy, the patient complained of a sore throat, and a laryngeal examination three days later showed congestion of the epiglottis with acute ulcerative laryngitis. On the

following day, the patient had difficulty in breathing, accompanied by choking, smothering and coughing. These symptoms ameliorated somewhat, but ten days later she suffered from shortness of breath, grew progressively worse, and was placed in an oxygen tent. A tracheotomy became imperative and was performed at midnight.

Present Condition: The patient consulted one of us (I) one year after the tracheotomy (March 29, 1938). She was wearing a well-placed tracheal cannula, and was unable to breathe without it.

Laryngeal Examination: The vocal cords were intact, of normal appearance, and near the mid-line. The right arytenoid cartilage moved slightly on attempt at phonation, while there was mere twitching of the left arytenoid. Below the vocal cords, the larynx was completely occluded by scar tissue which, on sounding from below, had a very firm consistency.

The patient was advised to have a laryngofissure performed, but up to the present time has not consented to operation. The lateral roentgenogram, Fig. 1, showed complete subglottic stenosis.

Comment: In this case laryngeal involvement, apparently secondary to perichondritis, became manifest three weeks after laparotomy and two weeks after the removal of an indwelling gastric feeding tube. The laryngeal stenosis required tracheotomy. One year later a complete fibrous subglottic diaphragm was manifest. Laryngofissure was indicated, but the patient refused operation. The friction of the duodenal tube must have initiated the perichondritis, which may have been aggravated by postoperative debility and sepsis. The surgeon[10] who performed the abdominal operation has had a vast experience with duodenal tubes, and stated that this is the first case of the kind that he has encountered.

CASE 3.—C. S. (No. 4324), white boy, age 4, was admitted to the Indianapolis Methodist Hospital on April 16, 1937. A laparotomy was performed the same day for a gangrenous appendix. A marked febrile reaction was present on admission. A No. 12 Levin tube was anchored April 26, 1937, for suction and irrigation. On April 27, 1937, a Witzel enterostomy was done with the Levin tube still in place. The tube was indwelling for eight days. Two days prior to the removal of the tube the patient complained of pain on swallowing, sore throat, paroxysms of coughing, with occasional cyanosis and dyspnea. The dyspnea and cyanosis gradually grew worse until on May 15, 1937, twelve days following the removal of the tube, it became necessary to do a hurried tracheotomy.

Fig. 1—Case 2. Roentgenogram showing complete subglottic stenosis of the larynx, 1-2; 3, tracheal fistula.

Laryngeal Examination: Direct, laryngoscopy immediately following the tracheotomy, revealed intense inflammation of the larynx with a marked subglottic swelling of the soft tissues. The pyriform sinuses were intensely inflamed with a marked edema around the arytenoids and the glottic chink.

Present Condition: The patient was examined April 26, 1939, by one of us (M) and was still wearing the tracheal cannula. Indirect laryngoscopy revealed the vocal cords in the paramedian position with only a slight movement of the arytenoids. A No. 8 laryngeal dilator could be passed through the subglottic stenosis. Patient is still under dilatation treatment.

Comment: In this case perichondritis became evident thirty days after laparotomy and five days following the removal of the indwell-

ing feeding tube. The first symptoms of laryngeal involvement developed two days prior to the removal of the feeding tube. The laryngeal stenosis required tracheotomy. Two years later the child is still wearing a tracheal cannula. Various attempts to relieve the stenosis have been made during the last two years and only recently has dilatation been effective. The friction of the feeding tube, aggravated by sepsis, must have been the cause of the perichondritis. (Wassermann test negative.)

CASE 4.—Mrs. J. W. (No. 8054), aged 40, was admitted to the Methodist Hospital, Indianapolis, May 23, 1937. A laparotomy was performed for a gangrenous appendix on the same day. A Levin tube No. 14 was anchored for suction and drainage following the operation. Two days later the patient complained of severe soreness in the throat and around the tube, and this continued for several days. On June 1, 1937, she complained of shortness of breath and vomited blood-tinged fluid. On June 5, 1937, the Levin tube was removed. At this time more vomiting was noted. On June 7, 1937, the tube was re-anchored for relief of distention and removed again on June 8, 1937. At this time a high febrile reaction was present. The patient was dismissed July 1, 1937, and was readmitted as an emergency case on July 8, 1937, because of severe dyspnea, necessitating a tracheotomy.

Direct Laryngoscopy: July 28, 1937 (twenty days later), revealed intense inflammation of the hypopharynx and pyriform sinuses. There was extensive edema over the arytenoids and glottic chink. The vocal cords were not visible on account of the edema. Patient was discharged wearing a tracheal cannula.

Present Condition: April 26, 1939, patient still wearing the tracheal cannula. She has been under treatment with metal laryngeal dilators for the past two years.

Comment: In this case irritation of the larynx and throat became manifest two days following the anchorage of a Levin tube. The tube was worn for fifteen days. Forty-seven days following laparotomy and thirty days following removal of the Levin tube, it was necessary to do a tracheotomy because of stenosis of the larynx. This was a very septic case. It is evident that the friction and pressure of the feeding tube on the cricoid cartilage in the presence of sepsis initiated the perichondritis with resultant stenosis of the larynx. (Wassermann test negative.)

CASE 5.—D. G. (No. 9263), white, male, aged 39, was admitted to the Methodist Hospital, Indianapolis, Aug. 3, 1937. A laparotomy was performed the same day for acute gangrenous appendicitis. A Levin tube was indwelling for eight days. Hoarseness and odynophagia developed while the tube was in place. On August 21, 1937, the patient had an additional operation for pelvic abscess. No feeding tube was used at the second operation. Patient was dismissed from the hospital Sept. 3, 1937. The hoarseness which developed while the tube was in place continued for two months following removal. The odynophagia continued for only one month. Stenosis did not develop and a tracheotomy was not necessary.

Laryngeal Examination: Indirect examination of the larynx one month prior to the first operation showed the larynx to be normal. Laryngeal examination by one of us (M) two months following removal of the Levin tube revealed the larynx inflamed with a moderate edema over the arytenoids. The vocal cords were decidedly red. There was some edema around the base of the epiglottis and a moderate thickening in the subglottic region, but there was no fixation of the arytenoids.

Present Condition: March 12, 1938, indirect laryngeal examination showed the larynx normal.

Comment: This patient had a laryngeal complication following the wearing of a Levin tube for eight days. The laryngeal obstruction was not severe enough to call for tracheotomy. Three months later he was still hoarse. At the end of six months the larynx had returned to normal. No other cause could be found for the laryngeal complication other than the friction of the feeding tube. (Wassermann test negative.)

CASE 6.—O. V'B. (No. 11377), white man, aged 22, was admitted to the Methodist Hospital on September 19, 1937. A laparotomy was performed the same day for a gangrenous appendix. A Levin tube No. 14 was anchored following the operation for suction and irrigation. There was a marked febrile reaction on admission and for several days following. The Levin tube was indwelling for eight days. No soreness of the throat or discomfort on swallowing was noted while wearing the tube. The tube was removed September 27, 1937. Five days after the tube was removed the patient complained of dyspnea which increased in severity for ten days, when it became necessary to do a tracheotomy. Dysphagia developed following the tracheotomy, and a Levin tube was re-anchored for

feeding only. The tube was indwelling this time for twelve days. The tube was removed the second time on October 26, 1937.

Indirect laryngoscopy by another laryngologist at this time reported a perichondritis with abscess of the thyroid cartilage, which was draining into the trachea.

Present Condition: The patient is still wearing a tracheal cannula. A laryngeal examination by one of us (M) March 29, 1939, disclosed a moderate fixation of the arytenoids, the vocal cords intact and in the paramedian position. The subglottic soft tissues are very dense and fill the entire lumen. Notwithstanding the frequent dilatations with laryngeal dilators for a period of eighteen months, the subglottic structures collapse completely when the dilator is withdrawn. Dilation with rubber core molds is now being considered.

Comment: In this case the feeding tube was indwelling for eight days. There was no discomfort noted while wearing the tube, yet five days following removal of the tube, dyspnea developed. Ten days later tracheotomy was necessary. It is evident that the friction of the feeding tube on the cricoid cartilage in the presence of sepsis initiated the perichondritis with resultant stenosis of the larynx. (Wassermann test negative.)

CASE 7.—M. C. (No. 89839), a colored woman, aged 28, was admitted to the Cincinnati General Hospital March 19, 1938, with a perforating bullet wound of the abdomen. On March 20, 1938, laparotomy was performed by Dr. Longacre. Six perforations were found in the lower ileum and six inches of bowel was resected. A Wangensteen tube was inserted through the nose and suction drainage applied. The tube was indwelling for six days, when it was removed. The patient recovered from the laparotomy without infection, and was out of bed on the eleventh postoperative day. During the sojourn of the tube she became hoarse, and after its removal she gradually developed respiratory difficulty associated with sore throat, slight inspiratory stridor, and occasional croupy cough. At times bloody mucus was seen in the gargle fluid. By the eighteenth day after the removal of the duodenal tube, the respiratory difficulty had become very severe and was associated with cyanosis and inspiratory stridor, accompanied by retraction at the sternal notch and in the supraclavicular spaces.

Upon laryngeal examination (second within two days) the epiglottis and vocal cords were found normal in appearance. Abduction and adduction were unimpaired. The right aryepiglottic

fold was red and edematous, with about twice the normal thickness. The left fold was congested, but not thickened. There was swelling and edema over the cricoid cartilage posterior to the arytenoids. The interarytenoid space was inflamed and edematous. This swelling seemed to be continuous with two sausage-like edematous folds, one beneath each vocal cord, leaving an interspace of about five millimeters.

Tracheotomy: Immediate tracheotomy, under local anesthesia, was performed by the house physician. When the trachea was opened, the patient ceased breathing. The trachea was aspirated, but breathing did not return. A bronchoscope was then inserted into the trachea through the tracheotomy, and artificial respiration was started. Carbon dioxide and oxygen were given through the bronchoscope. At the same time intracardiac adrenalin was administered. After about ten minutes, spontaneous respiration returned.

Following the tracheotomy, subcutaneous emphysema and signs of mediastinitis developed. The patient was again referred to the surgical department, and was twice operated upon with the hope of draining the mediastinum, but the patient succumbed within a few days.

Necropsy (by Dr. Ventress, coroner's physician): The right chest cavity was filled with blood and the right lung was completely collapsed. An abscess cavity, filled with pus and blood clots, was present in the posterior mediastinum, and the abscess had burrowed into the right chest cavity. In addition there was recently organized obliterative pericarditis. The esophagus, trachea, and larynx were removed together. On the anterior wall of the upper end of the esophagus slightly to the left of the mid-line, there was a deep oval ulcer beginning just below the articulation of the left arytenoid cartilage and extending downward for about three centimeters and about one and a half centimeters in its widest diameter. The ulcer had sharp edges throughout, and was deepest in its upper half, where it extended through the mucosa and muscle down to the perichondrium on the posterior aspect of the cricoid cartilage. (See Fig. 2.) The ulcer spread laterally along the cricoid cartilage on both sides, more on the left, giving rise to a deep abscess filled with brownish, necrotic material, and communicating with a mediastinal abscess. There was a round perforation about one-quarter inch in diameter in the anterior wall of the trachea about six inches below the glottis. The enteric anastomosis was intact without any evidence of peritonitis.

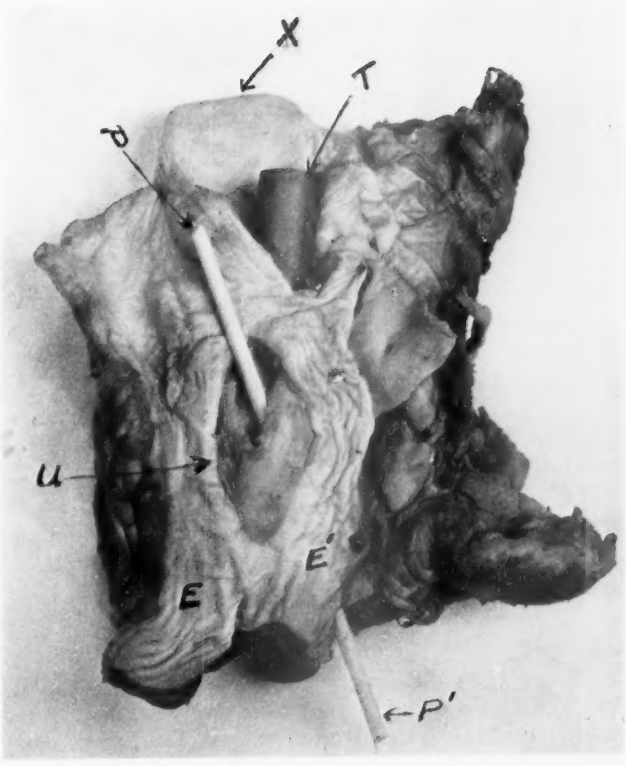

Fig. 2—Specimen from Case 7. The upper end of the esophagus, *E-E'*, is laid open from behind, revealing an oval ulcer in the long axis of the esophagus. The ulcer, *U*, extends through the mucous membrane and muscular coats down to the perichondrium on the posterior aspect of the cricoid cartilage. The white probe, *P-P'*, extends through the floor of the ulcer and into the mediastinum on the right. The rubber tube, *T*, is inside the larynx. *X*, the epiglottis.

Microscopic Diagnosis (by Dr. P. Wasserman): Section was taken in a horizontal plane at the level of the cricoid cartilage. Portion of the esophageal wall with recognizable mucosa was present on the posterior wall. An irregular inflamed defect with necrotic tissue was seen at one point apparently representing a penetrating esophageal ulcer. The inflammation extended along the regional fibromuscular tissue, approaching the outer surface of the cartilaginous ring. The cartilage presented considerable irregular, mottled, lavender, staining suggesting degeneration, possibly due to the regional soft tissue inflammation. The perichondrium was not made out here. Essentially the same type and degree of inflammation was seen in the soft tissue along the inner surface of the cartilaginous ring. The laryngeal submucosa, and in places the wall itself, was also inflamed. The picture suggested an acute ulceration through the entire thickness of the esophageal wall with extension of the inflammation along the soft tissue about the regional cartilage. The presence of the same type of inflammation on both sides of the cartilage suggested an extension of inflammatory reaction around the cartilaginous substance, possibly in the line of the perichrondrium (Fig. 3).

Summary and *Comment:* This patient wore an indwelling duodenal tube for six days following a "clean" laparotomy. She gradually developed signs of laryngeal obstruction which finally became so severe that tracheotomy became imperative eighteen days after the removal of the duodenal tube. When the trachea was opened, a prolonged period of apnea ensued. The trachea was first aspirated and then a bronchoscope was introduced through the tracheotomy and artificial respiration was employed, together with intracardiac adrenalin. Respiration and pulse finally returned. (The rare occurrence of prolonged apnea after tracheotomy has been described by Negus[14] in a recent monograph.) The patient developed mediastinitis from which she succumbed. Autopsy revealed a deep ulcer with perichondritis on the posterior aspect of the cricoid cartilage with extension of the inflammatory process into the lumen of the larynx (Fig. 2). In addition, the trachea had been perforated by the bronchoscope. This regrettable accident must have occurred during the attempt at artificial respiration with the bronchoscope in situ. At autopsy, it could not be determined definitely whether the mediastinitis which was found was secondary to the perforation or to the perichondrial abscess, although the latter seemed the more likely. This case and Case 8 are the only ones in our series in which autopsy findings have been recorded.

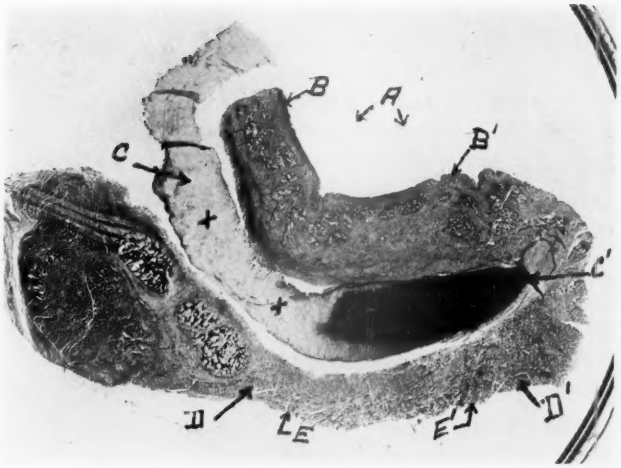

Fig. 3—Case 7. Cross section through the ulcerated esophagus and the cricoid cartilage at the level of the arrow, *U*, in Fig. 2. *A*, lies within the lumen of the larynx. *B-B'*, inflamed lining mucous membrane of larynx. *C-C'*, section of cricoid cartilage showing chondritis at *X-X*. *D-D'*, esophagus. *E-E'* ulcer in esophagus. It will be noted that the soft structures tend to separate from the cartilage (perichondritis).

CASE 8.—E. T. (No. 93036), aged 53, white, male, was admitted to the Cincinnati General Hospital April 15, 1938, in alcoholic stupor, with a penetrating wound of the abdomen. The patient refused operation. Generalized peritonitis soon became evident, and in spite of multiple transfusions and intravenous fluids, the patient steadily declined, and died on the tenth day after entering the hospital.

Treatment: A Wangensteen tube, with suction, was introduced, and remained in place almost continuously for nine consecutive days, after which time the patient pulled out the tube. The only signs which might have been attributed to the presence of the tube were occasional cough and expectoration of mucus, which more probably were due to the pulmonary complications. The larynx was not examined during the patient's stay in the hospital.

Necropsy: The larynx presented bright red, swollen, edematous epiglottic folds, with pinkish-white, markedly injected mucosa over the true and false vocal cords and an intensely hemorrhagic, granular,

lusterless mucosa just below the true vocal cords. At the superior end of the esophagus on the anterior wall in the region of the cricoid cartilage of the larynx there were three linear shallow ulcerations, varying in length from one to three centimeters and possessing shaggy edges and yellowish-gray bases. These ulcers varied in width from two to five millimeters.

Anatomical Diagnosis: Acute laryngitis, tracheitis, bronchitis and early lobular pneumonia superimposed on marked pulmonary congestion; acute ulcerative esophagitis and acute gastritis.

Microscopic Diagnosis: In spite of the autolytic changes that were present, there was evidence of recent ulceration substantiated by fibrinous exudate collected over a denuded area of the esophagus. (Dr. Reed.)

Comment: This patient wore a duodenal tube for nine days. Although the postmortem examination revealed ulceration of the esophagus at the cricoid level, with marked inflammatory reaction in the larynx, no striking laryngeal symptoms were noted in the history. The fact that the patient pulled out the tube may have some significance. This was one of the cases in which autopsy findings have been recorded.

Case 9.—A. H. (No. 26139), girl, aged 5, was admitted to the Indianapolis City Hospital on July 22, 1938, and was operated upon for a gangrenous appendix, with peritonitis, on the same day. A catheter was tied in the stump for cecal drainage. On the day after operation (July 23, 1938), a Levin duodenal tube, for suction, was introduced. The patient was very ill with peritonitis and received two blood transfusions, as well as oxygen inhalations. The Levin tube was indwelling for six days, after which time it was removed. One week later the patient became hoarse, with respiratory difficulty and dysphagia. The Levin tube was reinserted.

Because of increasing dyspnea, a tracheotomy was performed on the following day, and the Levin tube removed. Oxygen inhalations and another blood transfusion were given. There was a high febrile reaction for the next ten days. Laryngoscopy by one of us (M) about this time revealed intense inflammation of the larynx and pyriform sinuses with edema over the arytenoids and a closed glottic chink. The vocal cords were obscured by edema. An x-ray examination revealed soft tissue density of the subglottic structures.

Present Condition: On April 26, 1938, the patient was still wearing the tracheal cannula, and a direct laryngoscopic examina-

tion revealed the vocal cords intact, but immobile. The arytenoids also were immobile. The subglottic structures were collapsed, but the lumen admitted No. 26 rubber core mold.

Comment: After laparotomy for a gangrenous appendix with peritonitis, a suction duodenal tube was introduced and remained indwelling for six days. Six days thereafter dysphagia developed, with severe laryngeal obstruction which demanded tracheotomy. The patient still wears a tracheal cannula, and is being successfully treated by dilatation with rubber core molds. The septic condition of this patient, aggravated by the friction caused by the Levin tube, must have been a factor in producing the laryngeal perichondritis and resultant stenosis. (Wassermann test negative.)

CASE 10.—F. M., aged 19, female, white, was admitted to the Jewish Hospital, Cincinnati, September 22, 1938, with a previous history of congenital heart disease with mitral insufficiency and scoliosis. The patient was operated upon on the same day for papillary cystadenoma of the ovary at which time a bilateral salpingo-oophorectomy was done. At this time she also developed postoperative pneumonia, adynamic ileus, and probably localized peritonitis. Wangensteen suction drainage was employed for twelve consecutive days. The patient was discharged October 17, 1938, and readmitted October 25, 1938, with a history of vomiting. On November 3, 1938, the patient began to cough, had some dyspnea and air hunger with marked cyanosis. On November 4, 1938, the larynx was slightly tender; there was some subglottic swelling and redness.

X-ray examination of neck revealed a swelling of the soft tissues anterior to the fifth and sixth cervical vertebrae, with obliteration of the larynx lumen at and below the level of the vocal cords. "This was most likely due to an edema of the soft tissue below the vocal cords."

Tracheotomy: November 11, 1938. At this time dyspnea had become very marked, and tracheotomy under local anesthesia was performed.

Laryngoscopy: Subsequent examinations by direct laryngoscopy on December 6 and December 14, 1938, showed that the aryepiglottic folds were somewhat edematous. The vocal cords were also edematous and abduction was very much impaired, especially in the left cord. Immediately below the glottis there was an edematous mucosa which could be pushed aside. The trachea below this area was normal. On December 22, 1938, the vocal cords were still in-

flamed and thickened. The right cord was fixed in the midline. The left cord moved on adduction and abduction. Motion was somewhat impaired, and there was subglottic edema below the left cord.

Present Condition: At the present time the patient is still wearing the tracheal cannula and the laryngeal stenosis is being dilated.

Comment: This case presented the usual history of laryngeal inflammation becoming manifest some time after the discontinuation of suction siphonage through a duodenal tube. The laryngeal stenosis required tracheotomy. Some fixation of one arytenoid and subglottic stenosis are still present, and the patient is unable to dispense with the tracheal cannula.

Besides the cases cited above, we have collected two additional unpublished cases, as follows:

CASE 11.*—P. G., an adult male, was operated upon for acute appendicitis August 13, 1934. He developed a pelvic abscess, for which a secondary operation was performed on August 20, 1934. Wangensteen drainage was instituted September 2, 1934, for the relief of hiccough and distension. Three days later the patient began expectorating large amounts of green-colored sputum. About October 1, 1934, the patient's temperature began to rise, reaching a peak of 104.5 degrees on October 21st. "The chief symptoms during this episode were referable to the larynx, with hoarseness, pain, dyspnea and cyanosis."

Tracheotomy was performed October 23, 1934, and two weeks later the temperature had returned to normal.

After the inflammation had subsided, a considerable amount of obstruction persisted in the larynx. The laryngeal stenosis was treated over a period of two years, after which time the patient left the hospital and disappeared.

Comment: This was a very septic case. From the patient's record it cannot be determined just how long the feeding tube was employed. The laryngeal infection was accompanied by high fever. It is possible that the pyogenic infection of the larynx may have been metastatic in origin, and not necessarily due to the pressure of the duodenal tube, but this seems unlikely.

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*We are able to report this case through the courtesy of K. R. Nelson, P. A. Surgeon, U. S. Marine Hospital, Stapleton, N. Y.

CASE 12.*—*Abstract*—The patient, a white female, was ill with pneumonia for sixteen days (August, 1937). During this period the patient, because of unconsciousness, was fed via a Levin tube for about five days. After removal of the tube, the throat remained sore, with hoarseness and pain. Upon admission to the hospital, the larynx showed edema of the right aryepiglottic fold and the right arytenoid. There was fullness in the right pyriform sinus with the floor pushed up. An abscess formed and ruptured in the right arytenoid region, but tracheotomy was performed to relieve extensive subglottic edema. Tissue removed from the larynx showed chronic ulcerative pyogenic inflammation. A piece of cartilage was extruded. The laryngeal stenosis was treated by dilatation, and the tracheotomy tube was finally removed.

About three months after leaving the hospital, the patient committed suicide. The larynx was obtained and examination showed a web under the anterior commissure at the level of the cricoid cartilage. The area of ulceration had healed completely.

Comment: This is the only case in which the tube was employed for medical purposes. It is possible that the laryngeal perichondritis was secondary to the pneumonic process, but the patient attributed her trouble to the Levin tube, and this seems to have been the exciting cause. In an unconscious patient, injury to the larynx might easily escape attention. The pathological and autopsy findings were significant.

SUMMARY

Nine of the ten cases of laryngeal stenosis which we have encountered occurred in patients who had worn an indwelling duodenal tube after laparotomy. The tube was inlying for comparatively short periods varying from six to twenty days. The average period was eight and a half days. In most instances, the signs of laryngeal involvement were first recognized or became manifest after the permanent removal of the duodenal tube. The early symptoms of laryngeal or esophageal injury included pain, dysphagia, blood-streaked sputum, hoarseness, croupy cough, accompanied by more or less dyspnea. These symptoms gradually increased in severity over a period of days or weeks when dyspnea became the predominating symptom, and was so extreme that tracheotomy became imperative in all but two of the ten cases. Laryngeal examination in the acute stage revealed evidence of inflammation and edema. Sub-

*We are indebted to Dr. J. M. Lore, of New York, for the following case report.

glottic edema usually accounted for the extreme dyspnea. In the later cases, when the patients presented themselves permanently cannulated with tracheotomy tubes, various degrees of fixation of the arytenoid articulation, together with firm, fibrous, subglottic stenosis, were the predominating findings.

In two cases (Nos. 1 and 2), there was a complete subglottic occlusion. Five of the patients are still under treatment for laryngeal stenosis, and none of these patients has been able to dispense with the tracheal cannula. Two cases refused treatment.

Two cases came to autopsy. In one case (No. 7) there was deep ulceration of the upper end of the esophagus, with perichondritis over the posterior aspect of the cricoid cartilage and extension of the inflammatory process into the lumen of the larynx (Figs. 2 and 3). In Case 8 there were shallow linear ulcers in the upper end of the esophagus in the cricoid region associated with marked intralaryngeal inflammation.

COMMENT

From a study of these cases, it is apparent that the laryngeal inflammation was secondary to the presence of an indwelling duodenal tube. The primary lesion appears to be due to pressure necrosis of the esophagus at its attachment to the body of the cricoid cartilage. In this area over the median ridge of the cricoid cartilage, the muscular coat of the esophagus is very thin[15] and does not provide much of a cushion between the mucous membrane and the cartilage. From clinical experience (esophagoscopy), it is well known that the mouth of the esophagus is in a state of tonic contraction and that this contraction is only overcome when the cricopharyngeus muscle and the upper circular fibres of the esophagus relax. In other words, the circular muscle fibres,[16] together with the cricoid cartilage, constitute a sphincter at the entrance to the esophagus. This sphincter is unique inasmuch as it is made up of two parts, one muscular and the other cartilaginous. The constant pressure of the sphincter muscles on the duodenal tube would tend to force the latter against the cricoid cartilage, and this pressure, together with friction (swallowing, vomiting, irritating gastric juice, etc.), might lead to ulceration of the mucous membrane over the lamina of the cricoid. Furthermore, there may be pinchcock mechanism with pressure between the cricoid cartilage and vertebral column. If the ulceration extended to the deeper structures, perichondritis on the posterior aspect of the cricoid cartilage would ensue. From this area, the perichondritis could easily spread over the upper border of the cricoid

cartilage into its internal perichondrium, giving rise to acute subglottic stenosis of the larynx or to arytenoid fixation. The acute stage is followed by fibrosis with cicatricial stenosis of the larynx, in a manner similar to the effects of perichondritis arising from other causes. The only other explanation might be that the perichondritis is metastatic in origin, but it would appear strange for the larynx to become the only site for the lodgment of a metastatic abscess, and such metastases are not often recorded after septic laparatomy cases independent of the indwelling feeding tube. It may be true, however, that a septic condition of the patient may favor the formation of ulcers about the duodenal tube, especially since Mosher[17] has shown that superficial ulcers may occur in various portions of the esophagus in certain infectious diseases.

CONCLUSIONS

1. The indwelling duodenal tube has proven to be a boon to both patient and surgeon.

2. It is commonly stated that the indwelling tube may be worn with impunity and that no complications ensue from its presence.

3. However, on rare occasions ulceration of the upper end of the esophagus, complicated by laryngeal inflammation and stenosis, may arise from the presence of the tube. We have observed ten such cases, and have collected two additional cases.

4. The laryngeal stenosis is usually secondary to perichondritis of the cricoid cartilage.

5. Tracheotomy very frequently becomes imperative to relieve the acute laryngeal stenosis.

6. Subglottic fibrous stenosis often ensues, compelling the patient to become a chronic tube carrier.

7. In the presence of an indwelling duodenal tube any signs or symptoms indicating esophageal or laryngeal involvement demand immediate laryngoscopy.

8. The duodenal tube should be removed if the larynx or hypopharynx show any signs of inflammation.

9. Whenever feasible, the indwelling tube should be removed and cleansed at frequent intervals as a prophylactic measure. The tube should not be reinserted repeatedly through the same nostril.

10. It is advisable to employ duodenal tubes of small diameter.

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LXXII

SEPSIS FOLLOWING PHARYNGEAL INFECTIONS*

COLBY HALL, M.D.

LOS ANGELES

Severe sepsis is not an uncommon complication or sequel of pharyngeal infections, the true nature of which is too frequently recognized only at postmortem.

This paper deals neither with cavernous sinus phlebitis or meningitis in which the objective signs and the true nature of the process are usually evident, nor does it deal with submaxillary space infection or with that phlegmonous involvement of the floor of the mouth, usually dental in origin, known as Ludwig's angina. It is concerned, however, with general sepsis which has its origin in the pharynx or the pharyngomaxillary space, its extension by way of the internal jugular vein and tributaries, and in which the local signs are often very meager.

When one is confronted by such a case there are two essentials for its successful treatment: (1) A careful history of the case; (2) a knowledge of the anatomy and pathology of this region.

There are three routes by which infection may reach the general circulation from the pharynx: (1) Phlebitic; (2) phlegmonous; (3) lymphatic. Either one or a combination of these may be active in the individual case.

Let us first consider the phlebitic route (Fig. 1). The internal jugular vein receives blood from the pharyngeal and tonsillar plexus, usually by way of the common facial vein, although occasionally the anterior facial, the posterior facial, and a few small pharyngeal veins may empty independently into the internal jugular. A thrombophlebitic process arising in the small veins of the pharynx reaches the general circulation by the same route and, in addition, it may, after reaching the internal jugular, involve the lateral sinus by retrograde extension. Anatomically it seems quite

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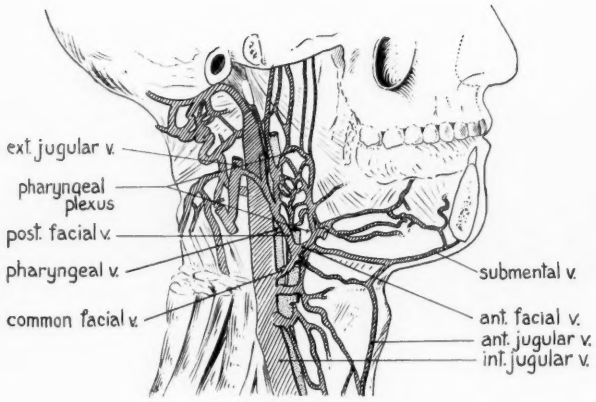


Fig. 1. Veins of the pharynx.

possible for the external jugular vein to become involved by way of the posterior facial or for the anterior jugular vein to become involved; however, I have not as yet encountered either condition clinically.

In consideration of the phlegmonous route we are mainly concerned with the pharyngomaxillary space (Figs. 2 to 7). The pharyngomaxillary space is a potential space separated from the tonsil, the pharynx and peritonsillar space only by the constrictor muscle and its fascial coverings. Posteriorly it is in relation with the contents of the carotid sheath; laterally the internal pterygoid, the parotid, and their respective fasciae form the boundary; while inferiorly it ends in a fascial cul-de-sac at the angle of the jaw. The pharyngomaxillary space is not connected with the retropharyngeal space—the firm alar fascia forming a complete separation between these two spaces. *It must be stressed again that the pharyngomaxillary space is a potential space and is actual only when made so by disease, by the anatomist, or by surgical exploration.* The pharyngomaxillary space is not infrequently the site of phlegmonous processes which may or may not eventuate in suppuration, and the internal jugular vein, situated as it is, may become involved in a periphlebitic or thrombophlebitic invasion. Arterial erosion and subsequent hemorrhage in pharyngomaxillary phlegmon is an uncommon but extremely dangerous complication to be remembered.

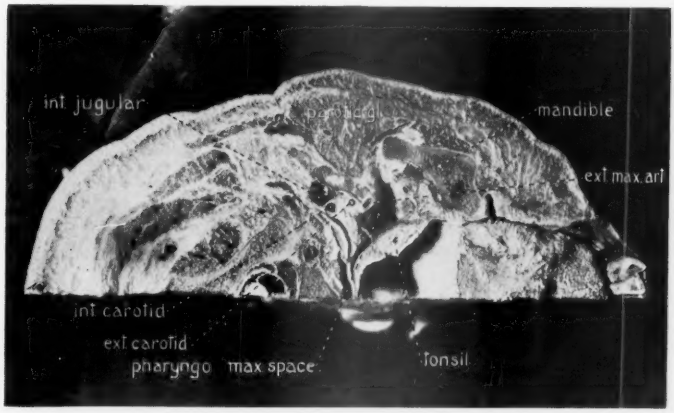


Fig. 2. Horizontal section of head at the level of the tonsil. The head has also been sectioned sagittally (the above photograph shows the left side only). The distended pharyngomaxillary space is clearly shown.

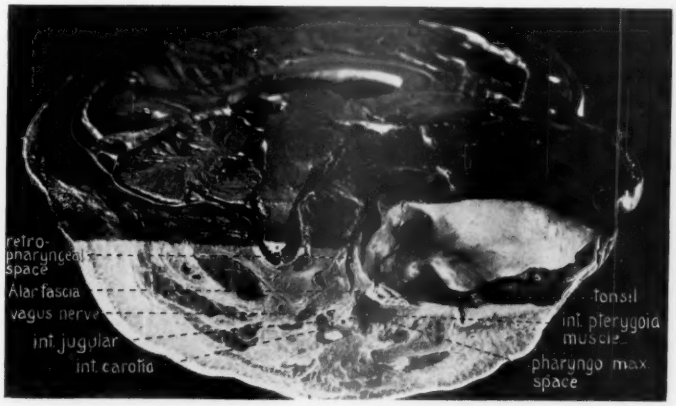


Fig. 3. Photograph showing the sagittal and the horizontal sections of the upper half of the left side of the head. The relationships of the pharyngomaxillary space are clearly shown.

The signs of pharyngomaxillary phlegmon are:

1. Extreme trismus.
2. Induration and swelling over the parotid and extending to below the angle of the jaw.
3. Obliteration of the "subangular space" beneath the angle of the jaw. This "subangular space" corresponds to the inferior boundary of the pharyngomaxillary space (Fig. 8).
4. Peritonsillar swelling and induration with little or no pharyngeal inflammation.

Because of the definite inferior fascial boundary, fluctuation occurs dangeroursly late if at all. A purely peritonsillar involvement displays pharyngeal and tonsillar inflammation without the extensive external signs and with a lesser degree of trismus; while in a primary submaxillary space infection all of the signs are more marked anterior to the angle of the jaw.

The lymphatic route involving the perijugular lymphatics has received much attention in the literature but has not been prominent in the experience of the author.

Clinically we recognize two types of cases:

1. Those in which sepsis follows immediately the pharyngeal infection.
2. Those in which the pharyngeal infection has subsided for from several days to several weeks, only then to be followed by severe sepsis.

Either type may be phlebitic or phlegmonous. In the phlegmonous type involving the peritonsillar or the pharyngomaxillary space, the diagnosis should present no difficulty. However, in the purely phlebitic type the pharyngeal signs are frequently so slight as to cause the fatal overlooking of the source of the sepsis. In these cases we must immediately rule out pyelitis, other pyemic foci, the possibility of erysipelas, endocarditis, blood dyscrasias, and true pneumonia and then be prepared to open the neck immediately. I say *true* pneumonia, because too often multiple lung abscesses resulting from the pyemic invasion have been confused with a true pneumonic process, the primary disease being entirely overlooked until too late.

In the presence of severe sepsis and the history of a recent pharyngeal infection, we are not justified in consuming an unrea-

Fig. 4. Photograph showing the sagittal and the horizontal section of the lower half of the left side of the head.

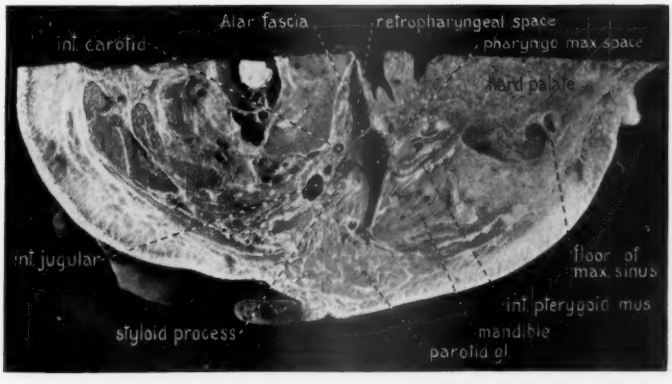


Fig. 5. Photograph of horizontal section at the level of the hard palate. Right side of the head. The distended pharyngomaxillary space is clearly shown. The alar fascia is very distinct and the lateral extension of the retropharyngeal space is demonstrated. Note the position of the styloid.

sonable length of time investigating rare diseases or performing various agglutination tests.

The hyperpyrexia, chills, and flushed appearance of the patient with acute follicular tonsillitis must not be confused with the septic temperature, severe chills, prostration, and pallid appearance of most of the cases herein described. This sometimes taxes our diagnostic ability.

The leucocytic count varies, but in the severe cases it ranges from 20,000 to 50,000, or even higher. The red cells and the hemoglobin tend to decrease as the disease progresses. Blood cultures are significant when positive.

The prognosis of the thrombophlebitic cases is dependent upon two factors: (1) The offending organism; (2) the duration of the disease before rational therapy is instituted.

We may say that in the cases due to the hemolytic streptococcus, and possibly the pneumococcus, the prognosis is better than when the invading organism is anaerobic, a staphylococcus, or the streptococcus viridans. The organisms of this latter group are more dangerous because of the metastatic abscesses which they form; the former group not having such a strong tendency in this direction. This explains why pharyngeal sepsis is usually more severe than that of otitic origin, the hemolytic streptococcus or the pneumococcus almost always being the offending organism in the otitic cases.

In the hemolytic streptococcus cases, repeated chills may occur without severe damage, and even late ligation may save the patient. This has been particularly true since the advent of sulfanilamide. However, since we have seen this drug mask a picture of otitic sepsis on the one hand, and cause a picture of sepsis on the other, we must constantly be on the alert during its administration.

In those cases in which anaerobic organisms, staphylococci, or the streptococcus viridans are the invading organisms, each successive chill signifies a more grave prognosis. In this type of case, if we could know the organism, the initial chill would be the indication for immediate surgical intervention. In one case of staphylococcic septicemia, sulfapyridine and staphylococcus phage were of no avail after the metastatic foci had become extensive. Early surgery followed by intensive chemotherapy is imperative in this group.

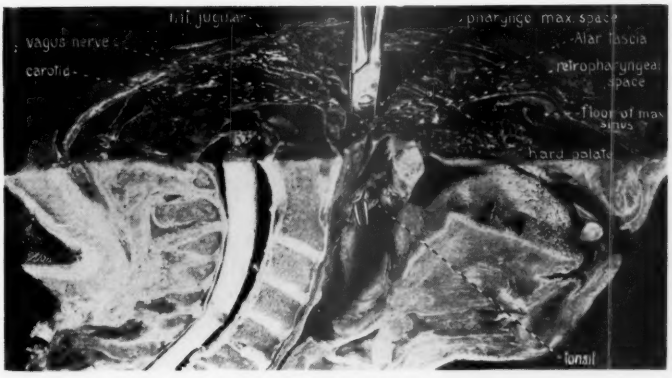


Fig. 6. Photograph of the sagittal and the horizontal section of the left side of the head at the level of the hard palate. Forceps are shown entering the distended pharyngomaxillary space from above and opening through the tonsil below. Again the alar fascia and the retropharyngeal space are clearly demonstrated.

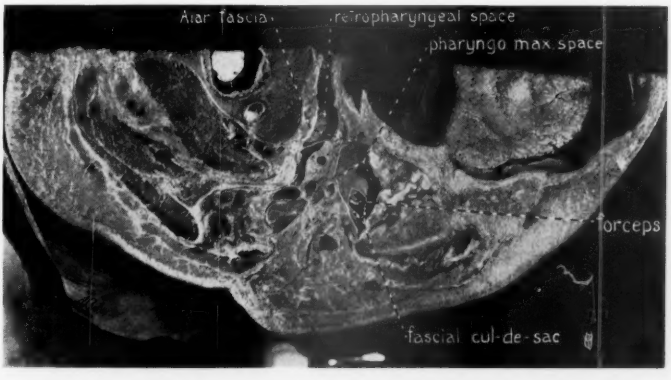


Fig. 7. Photograph of the horizontal section of the right side of the head. Looking from above downward. A forceps has entered the pharyngomaxillary space from below; the tips of the forceps are shown after breaking through the fascial cul-de-sac (inferior boundary) of the space at the angle of the jaw.

The prime requisites of treatment are: (1) To control the primary focus; (2) to cut off the primary focus from the general circulation; (3) to combat the metastatic foci.

To control the primary focus we must afford surgical drainage and, or, institute chemotherapy.

To cut off the primary focus from the general circulation, we must ligate the common facial vein, the internal jugular vein, or both. Ligation of the internal jugular must be made both above and below the entrance of the common facial vein, and beyond the area of the phlebitic process. A diseased vein should be resected, while resection of a grossly normal vein perhaps is optional.

To combat the metastatic foci we must rely upon chemotherapy and general supportive measures.

A case of peritonsillar or pharyngomaxillary suppuration in which chills or septic temperature occur or persist after adequate drainage or after subsidence of the inflammation should have venous ligation.

A pharyngomaxillary phlegmon not responding to chemotherapy in 48 hours and accompanied by sepsis should have external drainage of the pharyngomaxillary space and possibly venous ligation.

Any active or subsiding pharyngeal infection accompanied by sepsis should be considered as a thrombophlebitis, and this diagnosis proven or ruled out immediately. In case of doubt the carotid sheath and its contents should be investigated. Experience has proven that hours are precious in this condition and procrastination in instituting rational therapy is to invite a fatality.

In any case of unexplained sepsis the possibility of pharyngeal phlebitis must be considered.

When the common facial or the internal jugular vein is grossly diseased, both veins should be ligated. In case of doubt in a severe case, and where the veins appear normal, ligation should be done to block off the probable pharyngeal vein phlebitis from the general circulation. This has proven effective clinically in several cases. It has been suggested that in a case of this type it would be wise to ligate only the common facial vein. In two of my successful cases this would probably have sufficed and each patient would have been left with a patent internal jugular on the affected side. We must, however, remember that occasionally a few small

Fig. 8.

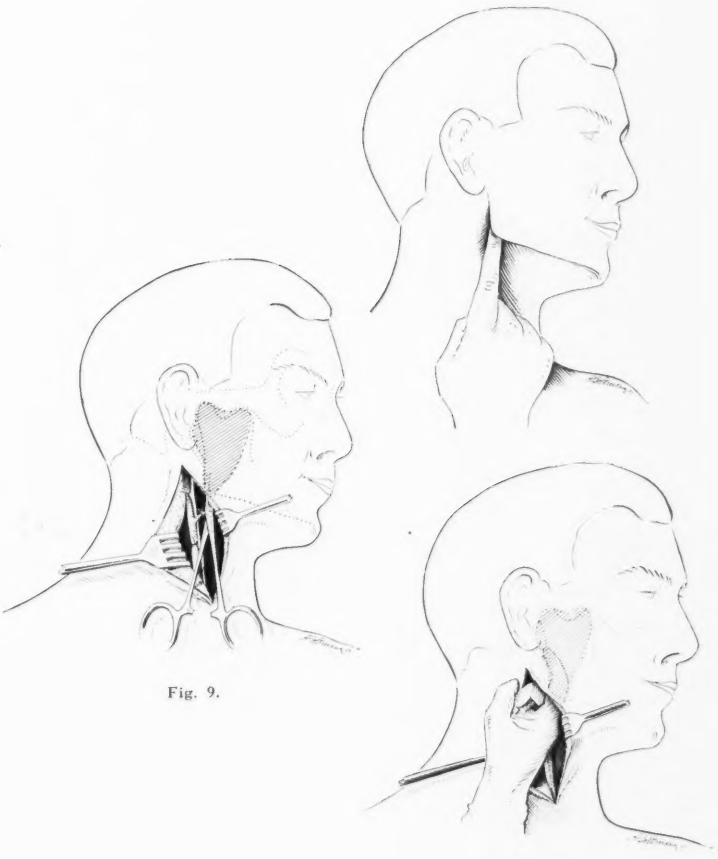


Fig. 9.

Fig. 10

Fig. 8. Subangular space. This indicates the inferior extremity of the pharyngomaxillary space.

Fig. 9. Diagram of approach to both the carotid sheath and the pharyngomaxillary space. Forceps are shown entering the space. The shaded area represents the extent of the distended pharyngomaxillary space.

Fig. 10. Finger is shown within the pharyngomaxillary space and palpating the important landmark—the Styloid Process.

pharyngeal veins empty into the internal jugular independently of the common facial vein.

In any case where the neck is opened the surgeon should investigate both the carotid sheath and the pharyngomaxillary space (Figs. 9 and 10).

Where retrograde thrombophlebitis has reached the lateral sinus a mastoid operation may be required.

The question may be asked, "How in a case in which the throat signs are vague or absent, do we determine which carotid sheath to expose?" The history of local pain in the throat or neck may be of great help. In my experience, even in the most difficult cases there has been some slight obliteration and tenderness of the "subangular space" on the diseased side. Tenderness along the course of the internal jugular vein has been seen only once, and that in a far advanced and fatal case.

Clinical and pathological variations can best be demonstrated by the following case reports:

REPORT OF CASES

CASE 1.—*Probable Phlebitis of the Pharyngeal Veins Following a Mild Pharyngitis.* G. E., white, male, age 21. Onset of illness, November 15, 1938. Patient had an upper respiratory infection which apparently cleared up, as he was symptom-free until December 4, 1938.

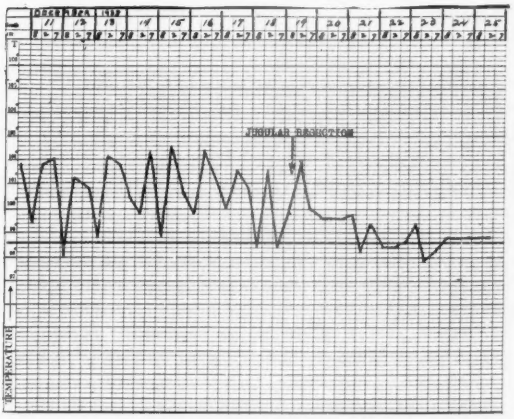
December 4: Slight soreness of the throat on the left. Temperature was normal.

December 6: Temperature rose to 102 degrees and reached 102 degrees or higher each day thereafter until admission to the hospital.

December 10: Admitted to the hospital.

Examination: The left lateral pharyngeal band was swollen, reddened, and covered with a grayish exudate. The tonsils had been completely removed.

Laboratory: R.B.C., 5,250,000; W.B.C., 10,400, 85 per cent polys. Culture from throat: hemolytic streptococcus, staphylococcus, and streptococcus viridans. Blood cultures repeatedly negative. The patient appeared very septic and had frequent chills and profuse sweats.



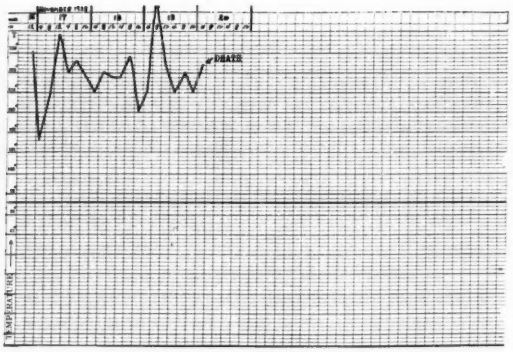
Case 1. Probable phlebitis of the pharyngeal veins following a mild pharyngitis.

December 14 to December 18: 375 grains of sulfanilamide were given with no apparent influence on the infectious process. Tenderness and slight obliteration of left "subangular space" persisted.

December 19: Venous ligation and resection. The left internal jugular and common facial veins were grossly normal. Both veins were ligated and resected. The pharyngomaxillary space was explored with no gross pathology being found.

Postoperative Course: Following surgery the patient made an uneventful recovery. The temperature returned to normal almost immediately.

Comment: This was evidently a phlebitis of the pharyngeal veins; the phlebitis feeding the general circulation but not having become apparent in the main radicals, namely, the common facial and the internal jugular. Because of the comparatively moderate nature of the illness, the absence of prostrating chills, the temperature not being in excess of 102 degrees, and no evidence of metastatic foci, it seemed justifiable to give chemotherapy a fair chance and to keep the patient under close observation rather than operating immediately.



Case 2. Thrombophlebitis of the internal jugular with a pharyngomaxillary abscess.

CASE 2.—*Thrombophlebitis of the Internal Jugular Vein with a Pharyngomaxillary Abscess.* B. M. H., white, female, age 15. Onset of illness, November 6, 1938. Patient developed a peritonsillar swelling and extreme pain on the right side of the throat.

November 11: Peritonsillar swelling "broke" with relief of local symptoms. However, high fever and extreme pain in the head, neck and limbs persisted.

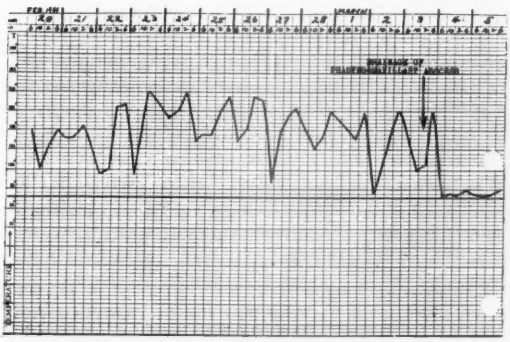
November 14: Parents noted that the child was becoming delirious.

November 16: Admitted to the hospital.

Examination: Right peritonsillar swelling. Extremely stiff neck. Upon admission the patient was irrational, was having a severe chill, and the temperature was 103 degrees.

Laboratory: R.B.C., 4,690,000, 78 per cent hemoglobin; W.B.C., 40,750, 92 per cent polys. Spinal fluid: 12 lymphocytes. Chemistry and pressure normal. Queckenstedt normal. Repeated blood cultures normal. Throat cultures: staphylococcus.

Hospital Course: A diagnosis of confluent pneumonia of the entire right lung and of the lower lobe of the left lung was made. For this reason the throat was given no more attention, and the laryngological service did not have an opportunity to see the patitent. In-



Case 3. Pharyngomaxillary (perijugular) abscess three weeks following a mild pharyngitis.

tensive sulfanilamide therapy, small transfusions and other supportive measures were of no avail.

November 20: Patient died.

Postmortem: Pharyngomaxillary abscess, right. Thrombophlebitis of right internal jugular and tributaries. Multiple lung abscesses. No intracranial pathology. Cultures from neck and lungs: staphylococcus.

Comment: Procrastination at home sealed the doom of this girl. After entrance to the hospital the pulmonary and meningeal signs so confused the picture as to cause the true nature of the process to be overlooked. It is improbable, however, due to the late stage at which this patient was seen, that surgery would have been of value.

CASE 3.—*Pharyngomaxillary (Perijugular) Abscess Three Weeks Following a Mild Pharyngitis.* J. E. R., white, male, age 38. Onset of illness, January 30, 1934. Patient had a mild upper respiratory infection associated with a sore throat. This had entirely cleared up by January 28.

January 28 to February 14: Patient was symptom-free.

February 14: Beginning soreness in the left neck. This soreness became progressively worse, and by February 19 there was

marked external swelling of the left side of the neck and face associated with fever.

February 19: Admitted to the hospital.

Examination: Classical picture of a pharyngomaxillary phlegmon. The tonsils had been cleanly removed.

Hospital Course: The patient continued to be very septic. The laryngological service were not permitted to treat the case.

February 24: An inadequate superficial incision was made over the swollen area. The operator did not wish to do even this because of the absence of fluctuation. Roentgen ray and foreign protein therapy followed.

March 3: No fluctuation had yet appeared and the sepsis continued. The patient at this period was very ill. Upon the patient's insistence a deeper exploration was made. There was an escape of over one ounce of foul-smelling pus.

Postoperative Course: Following adequate drainage the sepsis promptly ceased and all pharyngomaxillary signs disappeared. There was a long period of convalescence, however.

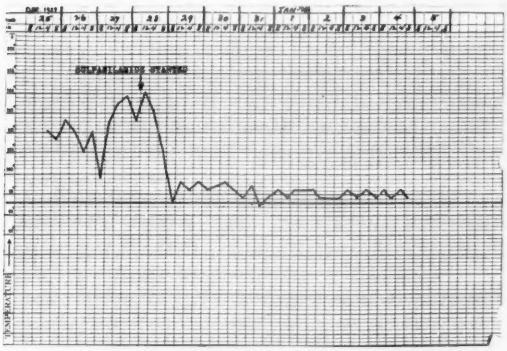
Comment: This was a case of perijugular abscess. The pharyngomaxillary space should have been surgically drained very early. The folly of awaiting fluctuation in this type of case is clearly demonstrated. It was indeed fortunate that the patient's resistance was superior to the organism's virulence. Procrastination to the degree practiced in this case is too great a gamble to take.

CASE 4.—*Nonsuppurative Pharyngomaxillary Phlegmon Three Weeks Following a Mild Pharyngitis.* M. W. L., white, female, age 73. Onset of illness, December 1, 1937. Patient had an upper respiratory infection and a mild pharyngitis which persisted only a few days.

December 25: Patient noted a deep soreness in the left throat and neck.

Examination: A firm peritonsillar induration with no tonsillar or pharyngeal inflammation. Temperature 102 degrees.

December 26: Temperature remained at 102 degrees. Leucocyte count 25,000.



Case 4. Non-suppurative pharyngomaxillary phlegmon three weeks following a mild pharyngitis.

December 28: Examination on this day revealed a typical pharyngomaxillary phlegmon. Temperature reached 104 degrees. Sulfanilamide therapy started.

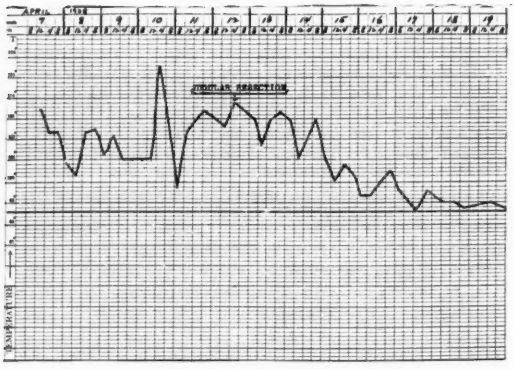
Course of Disease After Sulfanilamide: The temperature promptly returned to normal. By January 6, 1938, all evidence of sepsis and of pharyngomaxillary involvement had disappeared.

Comment: This was a nonsuppurative pharyngomaxillary phlegmon in an elderly diabetic. The response to chemotherapy was almost immediate. Had the sulfanilamide not been effective within 48 hours, I believe surgery would have been imperative.

CASE 5.—*Thrombophlebitis of the Internal Jugular Vein Following Immediately a Nonsuppurative Pharyngomaxillary Phlegmon.* B. M. B., white, female, age 31. Onset of illness, March 31, 1938. Patient had a sore throat beginning on March 31. This was associated with a swelling of the right side of the neck. Temperature rose to 104 degrees.

April 4: On this date patient had severe chills and temperature ranging from 103 to 105 degrees.

April 4 to April 7: Patient received 125 grains of sulfanilamide, but the severe chills and fever continued.



Case 5. Thrombophlebitis of the internal jugular following immediately a non-suppurative pharyngomaxillary phlegmon.

April 7: Admitted to the hospital.

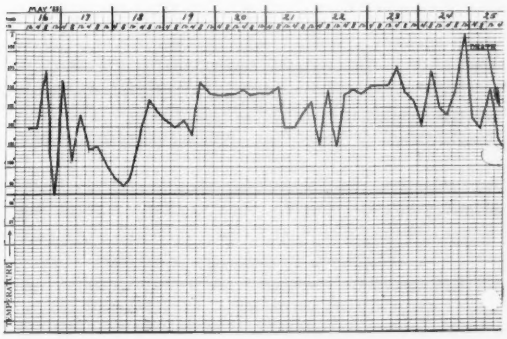
Examination: Tvpical right pharyngomaxillary phlegmon. The right tonsil and the uvula were edematous and pushed toward the left side of the throat, but there was no sign of pharyngeal inflammation.

Laboratory: R.B.C., 3,210,000, 63 per cent hemoglobin; W.B.C., 32,000, 94 per cent polys. Throat smear: only streptococcus. Blood cultures repeatedly negative.

April 10: Pharyngomaxillary signs had entirely disappeared and the temperature was receding. At 5:30 p. m. patient had a very severe chill and the temperature rose to 105.4 degrees and leucocyte count increased to 48,000. Blood culture taken at this time was negative.

April 11: The leucocytes were now 58,000 with 93 per cent polys. The red cells were 2,600,000 with 62 per cent hemoglobin. There was a slight obliteration and tenderness of the right "subangular space"; otherwise, nothing to guide to the diseased side of the neck. Pharynx was normal and all visible pharyngomaxillary signs had disappeared.

April 12: Venous ligation and resection. The right common facial and internal jugular veins were grossly diseased. Ligation



Case 6. Thrombophlebitis of the internal jugular following immediately a peritonsillar phlegmon.

and resection were done. The pharyngomaxillary space was free of gross pathology. Microscopic examination of the veins: acute thrombophlebitis.

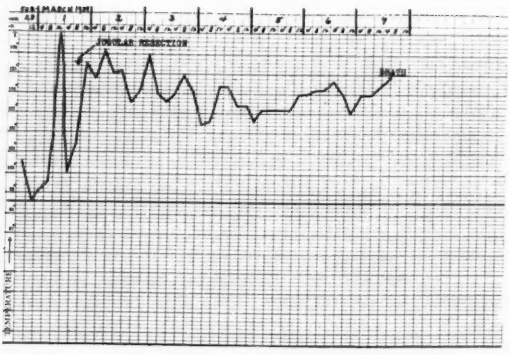
Postoperative Course: The temperature promptly returned to normal. Patient discharged cured on April 22.

Comment: This was a nonsuppurative pharyngomaxillary phlegmon which disappeared, leaving increased sepsis from a thrombophlebitis. To have waited because of the absence of pharyngeal signs would have been fatal. The type of streptococcus was not determined. This patient was fortunate that no metastatic abscesses had formed before the ligation and resection.

CASE 6.—*Thrombophlebitis of the Internal Jugular Vein Following Immediately a Peritonsillar Phlegmon.* L. L., Chinese, male, age 30. Onset of illness, May 14, 1938. Patient noted a sore throat, most marked on the left.

May 15: Admitted to the hospital. During the 24 hours prior to entering the hospital the throat pain had become much worse and the temperature had risen to 102 degrees. He also complained of some pain in the left side at the costal margin, both anteriorly and posteriorly

Incision of the peritonsillar swelling was followed by relief of local discomfort. No pus was recovered from the swollen mass.



Case 7. Thrombophlebitis of the internal jugular following immediately a mild pharyngitis.

On the evening of this day the patient had a sudden severe chill and temperature rise to 105 degrees.

Hospital Course: The chills continued. The temperature rose to 104 degrees and remained at about this level after May 19. The leucocytes increased from 9,300 upon admission to 34,000. Pharyngeal signs rapidly disappeared. Blood cultures: unidentifiable gram-negative bacillus and streptococcus viridans. All other laboratory procedures and physical examinations were negative. Sulfanilamide therapy was of no avail.

May 25: Patient died.

Postmortem: Thrombophlebitis of left internal jugular vein. Multiple septic infarcts of the lung. Most marked in left lower lobe.

Comment: This was a peritonsillar phlegmon with pharyngeal and tonsillar inflammation, little trismus, and no external signs. Again the pain in the side and the subsiding pharyngeal inflammation were deceiving. In retrospect, and at the present time, we would perform a venous ligation and resection which would be followed by sulfapyridine therapy.

CASE 7.—*Thrombophlebitis of the Internal Jugular Vein Following Immediately a Mild Pharyngitis.* C. O., white, male, age 24. Onset of illness, February 21, 1939. Patient noted a slight soreness of the throat for the first time.

February 24: Severe chills occurred for the first time. These chills continued each day until admission to the hospital.

February 28: Admitted to the hospital.

Examination: Pharynx and tonsils very mildly infected. So mild, in fact, that in the absence of history they would have been passed up as being normal. There was a moderate tenderness and obliteration of the "subangular space" on the left side. This was the only possible lead as to the diseased vein.

Laboratory: W.B.C., 26,400, 90 per cent polys.

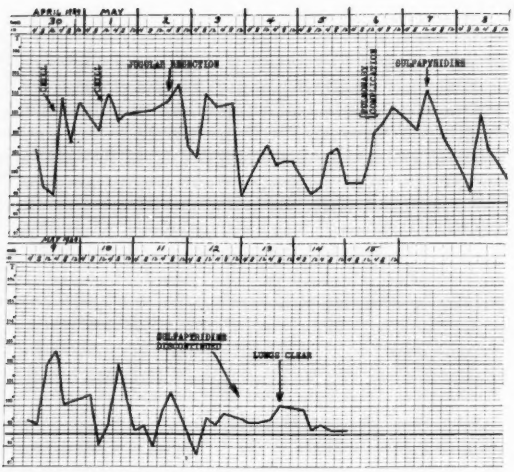
March 1: Temperature rose to 107 degrees, with dyspnea, cyanosis and pain in the left chest.

Radiographs: Diffuse patchy infiltration throughout the right lung and a smaller amount in the left lung. Venous ligation and resection. The left common facial and the internal jugular vein were markedly thrombophlebitic. The jugular phlebitis extending from above the facial to the clavicle. The pharyngomaxillary space contained no gross pathology. Microscopic examination of the veins: acute thrombophlebitis. Culture of the veins: staphylococcus aureus.

Postoperative Course: Patient was given sulfapyridine, transfusions, staphylococcus phage, and other supportive treatment but succumbed to his extensive pulmonary involvement.

March 7: Patient died.

Comment: This was a very mild pharyngitis which caused a rapid and extensive thrombophlebitis, staphylococcic invasion, and death in twelve days. The original source of infection was cut off from the general circulation at operation on March 1, but not until after five days of chills and the establishment of extensive metastatic foci.



Case 8. Probable phlebitis of the pharyngeal veins following a peritonsillar abscess.

Case 8.—*Probable Phlebitis of the Pharyngeal Veins Following a Peritonsillar Abscess.* H. C., white, male, age 41. Onset of illness, April 14, 1939. There was a very slight sore throat on this date. During the succeeding two weeks this became progressively worse, and a peritonsillar abscess formed on the left side.

April 28: Rupture of the peritonsillar abscess with the escape of foul, yellow pus. The local symptoms were promptly relieved.

April 29: Patient noted severe chills for the first time and these were associated with fever and intense headache.

April 30: Admitted to the hospital. Patient had received 210 grains of sulfanilamide before admission.

Examination: Subsiding left peritonsillar abscess. There was a slight cough with no evident pulmonary findings.

Laboratory: R.B.C., 3,900,000, 80 per cent hemoglobin; W.B.C., 14,000, 90 per cent polys. Blood cultures, negative.

Hospital Course: Patient had a chill on April 30 and another on May 1. All local signs were very mild. Radiograph of the chest was negative on May 2.

May 2: Venous ligation and resection. The left common facial and internal jugular veins were grossly normal. Both were ligated and resected. The pharyngomaxillary space was explored, but no gross pathology was found.

Postoperative Course: The temperature gradually declined and the patient improved. The leucocytes decreased to 10,000 and there were no more chills.

May 6: Temperature rose to 103.4, W.B.C. 30,400, pain in the left shoulder upon coughing. Radiographs: hazy acute consolidation of the upper one-third of the left lung. Sputum negative for pneumococci.

May 7 to May 13: 495 grains sulfapyridine and two transfusions.

May 13: Temperature normal; radiographs normal; patient clinically cured.

Comment: This was a subsiding peritonsillar abscess which left in its wake a generalized infection which sulfanilamide failed to influence. The venous ligation cut off the probable pharyngeal phlebitis from the general circulation. The pulmonary pathology was believed to be metastatic, the organism unknown, and responding to sulfapyridine. It is not difficult to speculate as to the outcome had prompt rational therapy not been instituted.

CONCLUSION

An understanding of anatomy, pathology and bacteriology is necessary for the prompt recognition, the early rational therapy, and the successful recovery of these cases of sepsis which complicate or follow pharyngeal infections.

Be not deceived by a comparatively innocent appearing pharynx, as the veins of this pharynx may be carrying the death sentence for your patient.

1136 WEST 6TH STREET.

LXXIII

INDICATIONS FOR DIRECT LARYNGOSCOPY*

CHEVALIER L. JACKSON, M.D.

PHILADELPHIA

Direct laryngoscopy dates from the 23rd of April, 1895, when Kirstein first employed a flat spatula illuminated by Casper's "prism hand lamp" to visualize the vocal cords. In this country, Fletcher Ingals of Chicago, Algernon Coolidge of Boston and Chevalier Jackson were pioneers in the development of this new procedure. Chevalier Jackson used at first the sitting position, as illustrated in his first book[1] 1907). Among Mosher's many contributions to the development of direct laryngoscopy, I might mention his interesting papers on "Direct Examination of the Larynx and of the Upper End of the Esophagus by the Lateral Route"[2] (1908), and "Direct Intubation of the Larynx"[3] (1909). Detailed consideration of the history of direct laryngoscopy may be found in the paper by Goldsmith[4] read before the American Laryngological, Rhinological and Otological Society in St. Louis in 1924, at its thirtieth annual meeting.

A full discussion of indications for direct laryngoscopy is found in a paper read by Fletcher Ingals[5] in 1911, also before an annual meeting of the "Triological" Society. Briefly, the indications he mentioned were: "impacted or imbedded foreign bodies in the larynx; laryngeal conditions in infants and young children, for diagnosis; inspection of parts of the larynx invisible or poorly seen in the mirror; and the evaluation of the nature limitation and extent of laryngeal lesions."

INDICATIONS FOR DIRECT LARYNGOSCOPY IN CHILDREN

Direct laryngoscopy[6] provides the only means of examining the larynx of infants and young children, and therefore the first indication for this procedure to be mentioned is the presence of any symptom referable to the larynx in a child. Obstructive laryngeal dyspnea, stridor, wheezing, hoarseness or aphonia call for direct laryngoscopy as the most important diagnostic procedure, and often the only means of treatment.

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*Presented before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, Mass., January 11, 1939.

Direct laryngoscopy in a large number of cases, by various individuals, has shown that the most common cause of stridor in infancy is congenital deformity of the epiglottis and supraglottic aperture. There may be true deformity, or simply an exaggeration of the Omega-shaped "infantile" epiglottis. This condition is rarely serious and is generally outgrown during the second year of life without treatment. However, it is extremely important to diagnose it and to exclude other conditions which do require treatment and may prove fatal if not treated. For example, congenital subglottic webs, papillomata, bilateral abductor paralysis, or dislocation of the aryt noids can be accurately diagnosed only by direct laryngoscopy.

Obviously, foreign body in the larynx of a child, as in an adult, constitutes an indication for direct laryngoscopic removal. Direct laryngoscopy affords a means of obtaining quickly and accurately specimens of secretions or exudate from the larynx and trachea for bacteriologic study, this being especially important in the differential diagnosis of laryngeal diphtheria and nondiphtheritic laryngotracheitis.[7] In asphyxia neonatorum direct laryngoscopy should always be done for aspiration of laryngeal and tracheal secretions and to exclude high obstruction as a cause.

Before leaving the subject of indications in children, I think it is of considerable historical interest to mention the fact that Dr. Max Thorner,[8] in 1899, chose as the title of his thesis for the American Laryngological Association, "The Direct Examination of the Larynx in Children." This is one of the first references to the subject in English, appearing only a few years after Kirstein's[9] original papers on "autoscopy," as he called it. Kirstein himself had written a paper on "Advances in the Laryngological Examination of Young Children,"[10] published in the Berliner Klinische Wochenschrift, and Escat, of Toulouse, had also written an article entitled "Direct Laryngoscopy in Children,"[11] published two years before.

It is interesting to note that Thorner used Kirstein's method in a series of cases "with good results," but he states, "only in extremely rare cases could I succeed in seeing the glottis!" Another series of children eighteen months to nine years of age was examined by Thorner with the Escat spatula, but he states that in this series also, he "rarely succeeded in seeing the glottis." Thorner believed that direct laryngoscopic methods would not be required when indirect laryngoscopic examination was "at all feasible," and he advocated it mainly in "very small children," but he concluded that the method constituted a step forward and a distinct advantage over our "former helpless inactivity" in certain cases.

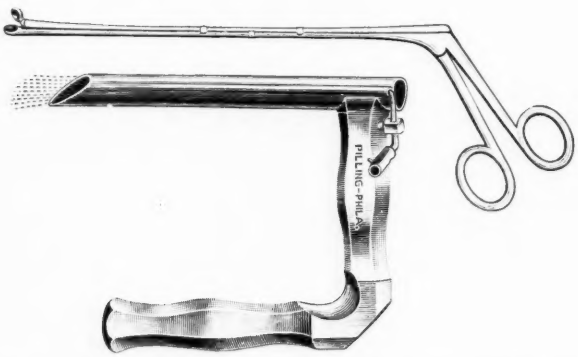

Fig. 1. Direct laryngoscope used for endolaryngeal operating, such as removal of benign tumors and the taking of tissue for biopsy (Chevalier Jackson anterior commissure laryngoscope, smooth-tipped model). The forceps illustrated are those advised for the removal of benign tumors.

INDICATIONS FOR DIRECT LARYNGOSCOPY IN ADULTS

The adult patients requiring direct laryngoscopy[6, 19] may be divided roughly into two classes, those in which the larynx is imperfectly visualized by the laryngeal mirror, and those in which the larynx is well visualized by the mirror, but some condition is revealed which calls for instrumental manipulation, such as biopsy, the removal of a benign tumor, or dilatation. The cases in which the larynx is not well seen by the mirror constitute a very important group, because if a hoarseness is allowed to persist without a complete view of the larynx having been obtained, clear up into the anterior commissure, there will be a great risk of overlooking an early carcinoma or other lesion urgently requiring treatment. To quote Chevalier Jackson,[12, 19] "Death often lurks under an overhanging epiglottis." An overhanging or deformed epiglottis is the most common cause of difficult visualization by the indirect method, though some other anatomical condition, or an exceptionally intolerant throat, may be at fault.

Before taking up the second group of cases, those in which the larynx can be well seen in the mirror, but a lesion is found which requires some instrumental manipulation, it should be stated that the indications as here given are simply the indications recognized in our daily work at the Temple University Clinic. In other clinics different indications may be followed; for example, New and Erich,[13] in their excellent paper on benign laryngeal tumors just published,

favor the use of the indirect method for the removal of small benign tumors and the use of suspension laryngoscopy for the larger ones.

Papilloma: Multiple papillomata, in adults as in children, are best treated, we believe, by repeated direct laryngoscopic removal, using a cupped forceps and taking care not to damage the underlying tissues. New favors electrocoagulation rather than avulsion with forceps. In cases where histologic study shows evidence of especially rapid growth, protracted fractional irradiation may be substituted, as advised by Foster.[14]

Vocal Nodules: This term is used strictly in reference to the tiny fibrous nodules which appear at the junction of the anterior and middle thirds of the vocal cords, especially in singers. There is some difference of opinion as to whether or not such nodules should be removed. I have obtained excellent results, in a number of cases, by direct laryngoscopic removal, and I think it is definitely indicated. Very careful accurate work is necessary, however, the cord being well exposed and firmly fixed, and then the nodule being neatly clipped off, without damage to the underlying normal fibers of the cordal margin.

Organizing Hematoma: The type of benign tumor variously called a "polyp," "benign tumor of inflammatory origin," and often erroneously called a fibroma, fibroangioma, or angioma, constitutes one of the commonest and most satisfactorily treatable laryngeal lesions encountered. Our pathologists have taught us to use the term "organizing hematoma" for these lesions, and we believe it is a very appropriate one, entirely consistent with their probable etiology and pathogenesis. Certainly the results of direct laryngoscopic removal are most satisfactory. As in the case of vocal nodules, it is necessary to scalp off only the abnormal tissue, leaving the cordal margin intact, if the best vocal result is to be obtained. It is better to remove the tumor superficially at first, and then to trim off the remaining abnormal tissue at a subsequent direct laryngoscopy, if necessary, rather than to remove the tumor completely at one time and risk damaging the underlying normal tissues.

Angiomas, fibromas and other true benign neoplasms do also occur; and they likewise call for direct laryngoscopic removal in most cases. If they are excessively large, thyrotomy, pharyngotomy, or partial laryngectomy may be required.

Polypoid corditis and *eversion of the ventricle* are conditions in which marked impairment of voice is produced, and while their

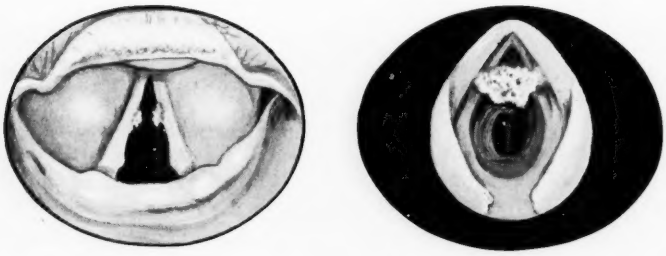

Fig. 2. Indirect laryngoscopic view (at left) of larynx of a young woman sent in for removal of what were thought to be two masses of papilloma, one on each cord; and direct laryngoscopic view (at right) of same larynx, showing that by this method the true character of the lesion was revealed—one growth in the anterior subglottic area, involving both cords just posterior to the anterior commissure. Biopsy proved the diagnosis of squamous cell carcinoma, and the tumor was successfully extirpated by laryngofissure. (Reproduced from color plate in "Cancer of the Larynx."[19])

treatment is not quite so satisfactory as that of more localized benign tumors and tumor-like conditions, generally a gratifying improvement in voice can be obtained. After a preliminary period of vocal rest and the reduction of all laryngeal irritation such as that of smoking and drinking alcohol, the patient is brought into the hospital, given a sedative, and his larynx carefully anesthetized. Then the larynx is exposed with the direct laryngoscope and the excess tissue carefully and accurately stripped off with cupped forceps. It is generally better to treat only one side at a time, having the patient come back in ten days or two weeks for operation on the other cord.

Contact Ulcer:[15, 16] When there are granulations present on a contact ulcer, and they do not disappear after a few weeks of vocal rest, it is of both diagnostic and therapeutic value to scalp them off by direct laryngoscopy. Healing is favored by the procedure, especially if a necrotic bit of cartilage comes away from the tip of the vocal process of the arytenoid; and histologic examination of the tissue will set the patient's and his doctor's mind at rest concerning the possibility of cancer, as well as excluding tuberculosis.

Laryngeal Tuberculosis: Direct laryngoscopic biopsy is sometimes advisable in tuberculosis of the larynx. Even if the patient has a frank pulmonary tuberculosis, it is not wise to take for granted that a laryngeal lesion is necessarily of the same character. If it is

at all atypical in appearance a biopsy should be done, because mixed lesions have been reported by Gabriel Tucker[17] and others. Cancer and tuberculosis may coexist in the same larynx, and in at least one of our cases these two diseases, with the addition of syphilis coexisted.[6, 19] Therefore, we do not refrain from doing a biopsy just because the patient is known to have tuberculosis or syphilis. Direct laryngoscopic cauterization and electrocoagulation have both been used with success by some laryngologists, but many prefer the indirect method for these procedures.

Carcinoma: Among the last and most important indications for direct laryngoscopy, let us mention again biopsy for the early diagnosis of carcinoma. No more urgent indication for direct laryngoscopy exists than the presence of a chronic hoarseness with slight roughening or thickening of the cord. By means of the smooth-tipped anterior commissure laryngoscope, even subglottic lesions can be visualized and specimens obtained for histologic examination. Certainly the earlier resort to direct laryngoscopic biopsy is now bearing fruit in bringing a larger and larger percentage of patients with cancer of the larynx to operation in time for surgical cure. Direct laryngoscopy affords also a means of definitely determining the gross extent of the lesion, and deciding upon its operability.

Direct laryngoscopic operations for the extirpation of cancer of the larynx are not to be recommended except in the rarest instances, but a few operators have succeeded in extirpating malignant tumors by suspension laryngoscopy.

The extraction of foreign bodies from the larynx, the dilatation of strictures, and the *introduction of the bronchoscope* are additional uses of the direct laryngoscope, though as for the latter, some prefer to pass the bronchoscope like an esophagoscope, without a laryngoscope. We prefer to use the laryngoscope routinely, but occasionally, in patients with a narrow protruding jaw, we have found it easier to pass the bronchoscope without it. As mentioned earlier in the paper, Dr. Mosher suggested the use of the direct laryngoscope for the introduction of intubation tubes, and he also reported a case in which he cut an adhesion between the cords by direct laryngoscopy preliminary to the introduction of the tube.

Asphyxial death may sometimes be prevented, in the drunken, injured or anesthetized patient, by lifting the tongue forward with the direct laryngoscope and thus eliminating the "lingual death zone," a catheter, intubation tube or bronchoscope being introduced, if necessary, to maintain the airway.

CONCLUSION

Not infrequently those of us who specialize in direct laryngoscopy and other peroral endoscopic procedures have patients sent to us with the request that we do a direct laryngoscopy. In such cases it is our practice always to examine the throat carefully with the tongue depressor and laryngoscopic mirror before deciding whether or not to make a direct examination. Quite often we find that by using particular care we are able to visualize the larynx very well by the indirect method, and to satisfy ourselves that the use of the direct laryngoscope is not indicated. In view of these facts, it seemed worthwhile to review, in this paper, the indications for direct laryngoscopy.

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LXXIV

EXPERIMENTAL STUDIES ON ENCHONDRAL BONE OF THE OTIC CAPSULE*

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Of the three bony layers of the human otic capsule, the enchondral layer is particularly deserving of attention and further study. This is because of its morphological peculiarities and the reaction of it to various pathological processes. Among the latter are included certain osteodystrophies, infections and fractures of the temporal bone. Our knowledge concerning this layer of the capsule has been based primarily upon experimental investigations, clinical observations and studies of serially-made sections through temporal bones.

It has been shown by Ulrich[1] that enchondral bone has no reparative powers and once fractured may offer an ever-present pathway for infections of the middle ear cavity to reach the labyrinth and meninges. It may become encroached upon or even replaced by pathologic bone of osteitis fibrosa and osteitis deformans (Nager and Meyer[2]). Of primary interest to the otologist is the otosclerotic process which in certain instances may involve the greater part of the capsule, the footplate of the stapes and even the ossicles (Covell[3]).

To study further the nature of the three layers comprising the otic capsule an experimental procedure has been utilized. Small fragments of bone from the region of the oval window margin of one rabbit were transferred to the anterior chambers of the eyes of other rabbits and changes observed in the transplants over a period of seven months. In this way it has been possible to ascertain the effects of depriving the bone of its blood supply, its changes when the vascularity becomes re-established and the ability of the transplant to form new bone. The results tend to confirm the clinical and laboratory studies for the human.

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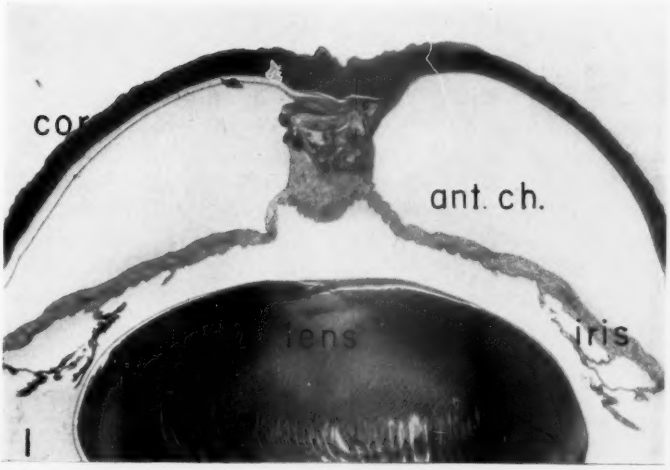
From the Laboratories of the George Williams Hooper Foundation, University of California, San Francisco, California.



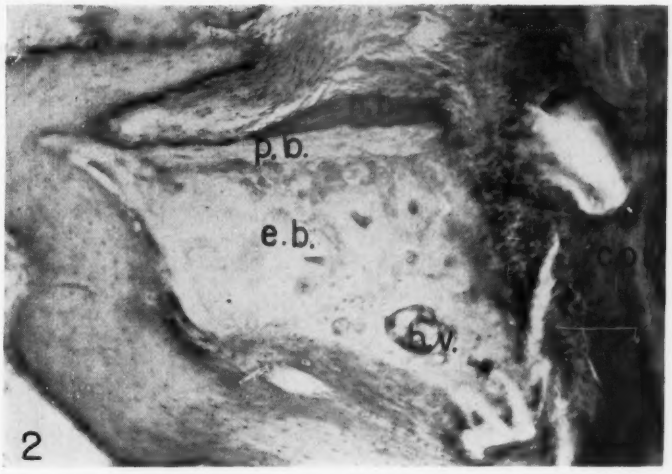


Fig. 1. Low-power view of a section through a 34-day-old transplant in the anterior chamber of rabbit's eye. The transplant is attached to the cornea as well as to outgrowths of vascular and fibrous tissue from the iris. The more deeply stained areas of bone represent new bone formation. (ANT. CH., anterior chamber; COR., Cornea.) Modified van Gieson stain, X12.

Fig. 2. Section through a 99-day-old transplant, showing a small area of new bone formation lying next to the necrotic periosteal bone. The enchondral bone stains feebly. There is no evidence of regeneration of bone in the vicinity of it. One large blood vessel and smaller vessels have a deposit of blue-stained substance around them. Hematoxylin and eosin, X160. (B.V., blood vessel; E.B., enchondral bone; N.B., new bone; P.B., periosteal bone.)

EXPERIMENTAL PROCEDURE

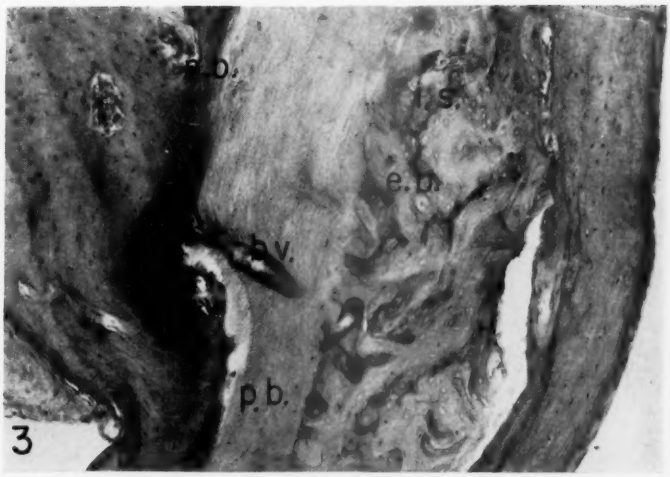
The anterior chamber of the animal's eye has been used by many investigators as a site for culturing various organs. More recently Bisgard[1] studied the effects of synthetic bone salts upon transplants of bone in the anterior chamber of the eyes of rabbits. In the present series of experiments the method used by Bisgard was followed. Small fragments of bone from the otic capsule were placed in a hypodermic needle, and after inserting the needle through the cornea in the neighborhood of the limbus, the contents of the needle were expelled into the anterior chamber by the stilet. Aseptic precautions were taken in each instance to assure no contamination of the transplant.

Sixteen rabbits were operated upon. Twelve of the transplants were successful and furnished the material for study. Four of the animals were sacrificed and their eyes removed after 33 to 47 days; three after the transplant had remained in the anterior chamber for 50 to 99 days; and five were allowed to remain until 155 to 218 days. The eyes were removed and the posterior half of the eyeball dissected and discarded, the remaining anterior half containing the lens, iris, cornea and graft was placed in Regaud fixative (80 parts, 2.5% potassium dichromate and 20 parts formalin, C.P.). After 24 hours the material was changed into fresh solution on four consecutive days and finally fresh daily changes of 2.5 per cent potassium dichromate for one week. Following the usual procedures of dehydration, the organ was imbedded in celloidin and sections of 10 micra in thickness were made. These were stained with either hematoxylin and eosin, a modified van Gieson or Schmorl's bone stain.

FINDINGS

While none of the material was examined microscopically before 33 days, it was evident that after seven to ten days the graft had started to grow. Vascular and fibrous tissue connections to the transplant were well established after 33 days (see Fig 1). There was evidence of new bone formation and necrosis of the old bone. The deeply stained substance about the bony fragments in the transplant illustrated in Fig. 1 is new bone arising next to the periosteal bone. The endosteal layer did not form new bone as did the periosteal layer.

Figure 2 represents a section through a 99-day-old graft which consists of periosteal and enchondral bone. Both layers stain poorly and the free surface of the periosteal bone has a thin layer of new



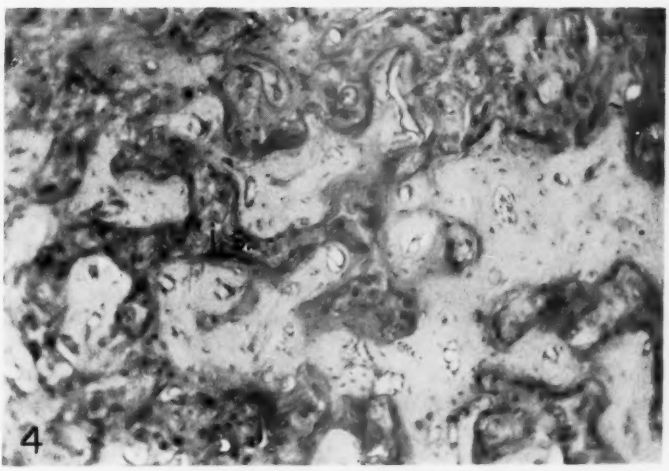


Fig. 3. Section through a 166-day-old transplant, showing features similar to that described for Fig. 2, with the exception that the contents of the interglobular spaces stain deeply and some of the nuclei of the calcified cartilaginous rests also stain. The blood vessel which passes through the necrotic periosteal bone to the enchondral bone is outlined by a heavier layer of blue staining material suggestive of the "blue mantles." (B.V., blood vessels; E.B., enchondral bone; I.S., interglobular spaces; N.B., new bone; P.B., periosteal bone.) Hematoxylin and eosin, X160.

Fig. 4. Enchondral layer of the intact otic capsule from an adult rabbit. The nuclei of the calcified cartilaginous rests stain deeply and the globuli ossei are in contrast to the interglobular spaces. Hematoxylin and eosin, X160.

bone attached to it. The whole transplant is surrounded by dense fibrous tissue on the lens side and is attached by fibrous tissue to the cornea anteriorly. The blood vessels within the substance of the necrotic enchondral bone are outlined by an intensely stained blue material. The cellular contents of the interglobular spaces and the globuli ossei are particularly indistinguishable at this stage.

A transplant which was allowed to remain in the anterior chamber for 166 days is shown in Fig. 3. The findings are similar to those of Fig. 2, with the exception that the interglobular spaces now stain and some of the nuclei of the calcified cartilaginous rests are also stained. A blood vessel which passes through the necrotic periosteal layer into the enchondral bone has a greater amount of darkly stained substance about it than is evident in the vessels of Fig. 2.

For comparison, the enchondral layer of an intact otic capsule of an adult rabbit is shown in Fig. 4. Here the interglobular spaces, their contents, and the globuli ossei are in contrast to each other. With the exception of the apparent vascularity of the enchondral layer of the rabbit's labyrinthine capsule, and the proportionately larger rests it may be said to bear considerable likeness to the human otic capsule.

DISCUSSION

In view of these experiments, it is of interest to review briefly certain features of the enchondral part of the human otic capsule as summarized by Nager.[5] (1) With the exception of the small ossicles of the middle ear (Oesterle[6]), this type of bone occurs in no other locality of the adult skeleton. (2) It is to be regarded as an arrested stage in the normal development of bone in cartilage. (3) It is completely formed in the newborn and its appearance is not materially changed throughout life. (4) It is poorly vascularized bone and its metabolism must be of an extremely low grade. (5) It does not participate in the usual processes of resorption, deposition and healing of bone.

It is to be recognized that many of these features for the enchondral bone of the human labyrinthine capsule may also be applied to similar bone in the capsule of the rabbit. However, it is to be remembered that the otic capsule of the monkey and dog bear a more striking resemblance to the human. The differences between enchondral bone and the small fragments of tibia used by Bisgard[4] in his experiments are more significant when we realize that he formed the following conclusion: "Bone, regardless of its viability,

has a favorable influence upon ossification." He found this to apply not only to partially viable bone and boiled bone but similarly to bone ash as well. It is obvious that enchondral bone must lack in its physico-chemical makeup the necessary combinations of calcium or phosphorous to influence ossification.

While the present experiments have no direct bearing upon the pathologic changes within the otic capsule for certain diseases, they serve to reveal many of its characteristics, as recently shown by Perlman.[7]

CONCLUSIONS

1. Enchondral bone of the otic capsule is incapable of forming new bone.

2. In young transplants it undergoes necrosis, while in older transplants there is some tendency for it to return to its usual appearance. The blood vessels in the latter are surrounded by mantles and the contents of the interglobular spaces are stainable.

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LXXV

DEEP NECK INFECTION*

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The findings in this paper are based on a clinical study of 78 cases of deep neck infection, and limited largely to the problems of etiology, symptomatology, diagnosis and management. The matter of surgical approach is limited to indication, without detailed descriptions of the technical aspects.

The incidence of age and sex in the entire series is as follows:

Age:

The youngest patient was eight months old, and the oldest eighty-one years, demonstrating that all ages are victims.

Under 2 years of age—8 cases ..... 10+%
Between ages of 2 and 12—37 cases ..... 47+%
Between ages of 13 and 19— 7 cases ..... 8+%
Between ages of 20 and 81—26 cases 33⅓%

Of infants and children there were 45 cases ..... 57+%
Of adolescents and adults there were 33 cases ..... 42+%

Sex: Of males there were 40 cases ..... 51+%
Of females there were 38 cases ..... 48+%

Regions of Involvement:

1. Pharyngomaxillary space infection—42 times ..... 53+%
2. Carotid sheath infection—11 times ..... 14+%
3. Submaxillary space infection—18 times ..... 23+%
4. Pretracheal fascia infection—8 times ..... 10+%
   (Buccopharyngeal—Visceral)
5. Prevertebral fascia infection—3 times ..... 3+%
6. Cervical lymph node suppuration—17 times ..... 21+%

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*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, 1939.

ETIOLOGY

This is regarded from two standpoints, the bacterial type of infection, and the portal of entrance. The bacterial incidence is as follows:

Streptococcus hemolyticus, pure culture	24 times
Streptococcus non-hemolytic	3 times
Streptococcus viridans	2 times
Staphylococcus aureus	3 times
Staphylococcus albus	1 time
Streptococcus hemolyticus and staphylococcus	1 time
Streptococcus hemolyticus, non-hemolytic staphylococcus albus and micrococcus catarrhalis in throat and streptococcus hemolyticus in neck	1 time
Streptococcus non-hemolyticus and staphylococcus aureus	3 times
Streptococcus hemolyticus, streptococcus viridans, staphylococcus hemolyticus and the fusi-spirochete organisms of Vincent's	1 time
Staphylococcus albus hemolyticus	1 time
Streptococcus hemolyticus in throat and pneumococcus type IV in neck	1 time
Streptococcus viridans and staphylococcus aureus hemolyticus	1 time
Streptococcus, not typed	3 times
Streptococcus hemolyticus, streptococcus viridans, micrococcus catarrhalis and staphylococcus aureus—from throat	1 time
Streptococcus, not typed, staphylococcus aureus and gram-positive diplococcus	1 time
Throat culture reported negative for diphtheria	2 times
Vincent's Angina organisms in tonsil and streptococcus hemolyticus in blood	1 time
Streptococcus viridans and staphylococcus albus	2 times

Staphylococcus, streptococcus and pneumococcus, not typed	1 time
Streptococcus and staphylococcus, not typed	1 time
Streptococcus hemolyticus and micrococcus catarrhalis	3 times
Streptococcus hemolyticus, streptococcus viridans and micrococcus catarrhalis in throat. No free pus in neck	1 time
Streptococcus non-hemolyticus and staphylococcus aureus non-hemolytic	1 time
Failed to grow (In one of these streptococcus viridans, staphylococcus aureus, micrococcus catarrhalis and Vincent's spirochetes were found in mouth	4 times
No culture obtained	15 times

None were cultured anaerobically. In some, direct smears were made from the mouth and pharynx.

Despite the fact that this evidence is incomplete, one is justified in concluding that the streptococcus hemolyticus appears in pure culture more often than any other organism, to the extent of about 40 per cent of those cases in which cultures were taken and grown; also that some form of streptococcus appeared in over 80 per cent of all the infections. This prevalence of the streptococcus coincides with Thomas' findings nearly thirty years ago in submaxillary infections (Thomas[2]).

PORTAL OF ENTRANCE

Knowledge of the portal of entrance has been very helpful to a full recognition of the pathway of subsequent infection, resulting in better management and a better conception of the prognosis. The tonsils and pharynx are the most common portals of entrance.

Entrance through tonsils and pharynx (includes adenoid)—53 cases	67+%
Of these, tonsils were absent in 10 cases and there were tonsil remnants present in 5 cases.	
Entrance following adenoid operation	1 case
Entrance following tonsil operation	2 cases
Dental infection following extraction	10 cases

Dental following filling of cavity and subsequent extraction of the tooth	1 case
Dental infection unprovoked	4 cases
Gingival following mouth trauma	1 case

The infection in one case (No. 14) followed the injection of a local anesthetic along the posterior surface of the maxilla. In one case (No. 4) the infection followed trauma to the mucosa of the hypopharynx by an unmasticated bread crust. One (No. 13) followed inflammation of the petrous part of the temporal bone. These were reported in a previous paper. There were thirteen cases with peritonsillar abscess classified here with those of tonsil origin (cases 39, 47, 48, 50, 51, 55, 57, 61, 65, 69, 70, 77 and 78). In one of them (No. 50) the abscess was not diagnosed before death, being found in the specimen removed at autopsy. One (No. 45) began in the lingual tonsil tissue at the base of the tongue. One (No. 37) followed the development of a deep ulceration and necrosis of the upper half of the tonsil caused by the fusi-spirochete organisms of Vincent's angina. This is also classified as of tonsil origin. In this case streptococcus hemolyticus was grown in blood cultures. One case (No. 15) previously reported as having been caused by an infected lymph node in contact with the submaxillary salivary gland, and for which no other cause was seen in the nose, pharynx or mouth, is now classified as of tonsil origin. Two years after the subsidence of the neck infection she returned for treatment because of a sore throat which was caused by an acutely inflamed tonsil remnant. This was later removed, since which she has had no further trouble. I now believe that the original infection of the neck in this case can be attributed to an inflammation of the tonsil remnant, which had subsided before she was seen. This remnant was in the lower part of the fossa, and it is possible that, because of its proximity to the side of the tongue base and floor of the mouth, the lymph drainage went by way of the lymphatics in relation with the submaxillary salivary gland, and caused infection there. To me it is noteworthy and somewhat startling that I have observed only one case of deep neck infection following or accompanying an acute exanthematous disease. In this instance (No. 56) it was scarlet fever. A retropharyngeal abscess would not be considered an unexpected event occasionally. A chronic sinusitis with recurring secondary attacks of acute pharyngitis has been noted in several instances (cases No. 24 and 26). One case (No. 66) was caused by an injury to the gum of the lower jaw when a playmate thrust a small stick into the mouth.

GENERAL SYMPTOMATOLOGY AND APPLIED ANATOMY

There will be only brief reference made to applied anatomy, and that of a simple explanatory nature pertaining to its influence on the behavior of the various types of infection. Respectful reference is made to the work of Mosher[3] which forms the basis of the anatomic application. In the succeeding chapters specific or characterizing symptomatology, diagnosis and management are stressed. For purposes of clarity and pertinency, references to management are carried along with the symptomatic and diagnostic narrative.

As a whole, unchecked neck infections go on to fatal termination by one or more of three main routes, viz., septicemia, asphyxiation or hemorrhage. With these end-results in mind it becomes desirable to make some attempt at classification in order to recognize early manifestations tending toward such involvements.

With regard to the symptomatology of neck infections, we are confronted with a great variety of manifestations, and a very considerable variation in the intensity of these manifestations. This makes classification difficult. In practically all severe fascial plane infections there is overlapping into one or more of these regions, but even in these the infection is predominant in some one of the regions, usually where it first involves the neck.

The visceral fascia extends from the skull base to the chest. The uppermost part of this, the buccopharyngeal fascia, where it covers the superior constrictor is the medial boundary of the pharyngomaxillary, or parapharyngeal, space. Here it is lateral to the tonsil. Lower and continuous with this it covers the middle and inferior constrictor muscles, thus limiting it below at the level of the lower part of the larynx or beginning of the trachea. This part is lateral to the hypopharynx and larynx. Lower where it envelops the trachea, esophagus and thyroid gland it becomes pretracheal fascia.

It should be remembered that this is all one continuous structure. A pharyngomaxillary infection is therefore at the start an infection involving the uppermost part of the visceral fascia. It is, however, not so designated until the lower parts of the visceral structure are involved, chief evidence of which is dysphagia, odynophagia, hoarseness and dyspnea, caused by involvement of the enveloped organs. Just behind the posterior part of the visceral fascia is the retropharyngeal space, the posterior boundary of which is the prevertebral fascia. It is then to be seen that when an infection encircles the pharynx from the parapharyngeal region laterally, the

retropharyngeal tissues will become involved. This is frequently to be seen in lateral neck radiographs, even without retropharyngeal abscess formation. In such instances, therefore, there is also present a prevertebral fascia infection. This is a rather frequent succession of events.

Suppuration in the pharyngomaxillary space is immediately in relation with the carotid sheath which extends downward through this space. Blood stream infection with or without thrombosis of the internal jugular vein is to be expected if such abscesses are not drained. Erosion of the internal carotid artery has also occurred (Salinger and Pearlman[23]). Large pseudoaneurysms form prior to final and fatal expulsive hemorrhage. A lymphatic jump of the infection from its starting point across otherwise uninvolved normal tissues into distantly situated lymph nodes, plants infection in that region.

High up in the neck three compartments of the cervical fascia are in close relationship with one another, the pharyngomaxillary, the parotid and the submaxillary. The last two are completely occupied by the respective salivary glands, the enveloping fascia uniting between the two glands. The pharyngomaxillary is a somewhat cone-shaped space, with its base uppermost at the base of the skull. Its apex is below at the hyoid bone and is continuous downward into the neck with the sheath of the great vessels. The retromandibular process of the parotid gland extends inward to reach the lateral side of the pharyngomaxillary space, and at this point there is often a defect or dehiscence in its fascia.

The prevertebral fascia encircles the vertebral column and the muscles attached to it. Above, it is attached to the base of the skull from which it extends downward into the chest.

It is very difficult to set forth an all-inclusive word picture of the symptomatology of neck infection because of the great variability of manifestations. Some symptoms are common to all types of neck infection, for which reason there will be a considerable repetition in the descriptions. No two cases are exactly alike, though there may be a great similarity in the cervical gland cases. The recognition of sepsis is of the utmost importance. Apathy, pallor, moist, sweaty skin surfaces, weakness, poor appetite and digestion, fever, especially with abrupt rises and drops, night or day sweating, septic blood count with increased young forms percentage, rapid, thready pulse of low volume, but perhaps full and bounding when the temperature is high, albumin and perhaps casts in the urine, and

chills. These are the common symptoms and signs of sepsis. A positive blood culture is of course conclusive. Where the word sepsis is used in this paper, such is the condition to which reference is made.

When it is recalled from which structures, symptoms and signs are derived, from the viewpoint of applied anatomy, the complexity is somewhat simplified. The structures giving rise to such symptoms are the lymphatic vessels and nodes, the blood vessels, the nerves, the cervical viscera and the muscles. From involvement of the lymphatics may be expected high continuous temperature, and this sometimes without visible swelling of the surface. From involvement of the veins may be expected high excursions of temperature and abrupt drops, with or without chills. Frequently there are periods of sweat drenching. Spasm or splinting of muscles occurs when the inflammation reaches their vicinity. The internal pterygoid and sternomastoid muscles are notable examples. There may be actual inflammatory pathology of the muscles (Coplin,[4] Discussion Thomas, and Case 56). Nerve involvement causes pain in the case of sensory nerves, and paralyses in the case of motor nerves. Pupils, pharynx, palate, tongue, larynx and shoulders may be thus paralyzed. Dysphagia, odynophagia, hoarseness, aphonia and dyspnea are indications of visceral involvement. A recognition of symptoms and signs resulting from involvement of these structures is therefore very helpful in locating the site of the infection, and it is also very helpful to know the exact portal of entrance. One may then anticipate the probable pathway of infection even when, as occasionally happens, there takes place a lymphatic jump, for the lymphatic drainage connections are fairly well known (Trotter,[5] Looney[6]). Often valuable clues are obtained from the behavior of the lymph nodes. To such symptoms and signs as are already present there may be added a lymph gland abscess. In other instances the glands may not produce an abscess, but may be the seat of severe acute inflammatory swelling, and if they are in relation with the carotid sheath, a jugular vein infection with or without thrombosis markedly increases the gravity of the disease by adding a blood stream infection. The lymph gland infection may result directly from the inflammation of the upper respiratory tract mucosa or tonsils, and thus constitute the whole phenomenon in the neck; or the gland infection may not occur until after the infection has already involved one or more of the neck regions by continuity. This is one of the chief sources of controversies among consultants, particularly when these are workers in different branches of practice. The pediatrist, over a short period of time, sees hundreds of cases

of cervical lymph gland inflammations, the vast majority of which subsides spontaneously. It is very natural for him, therefore, to temporize and advise accordingly. When, however, there is fluctuation the abscess should be drained even when there is no sepsis. In others fluctuation cannot be elicited, and these should be very carefully watched, because there may occur a sudden, severe sepsis which demands immediate drainage. A neglected gland abscess may also cause such an explosion, and result in a fatality. This I have seen. To the pediatrist neck infection too often means only cervical adenitis.

A number of interesting facts have been gleaned from a review of the blood counts in these cases. The total white count is almost always elevated, ranging in this series from normal to 39,500 (Case 63). The percentage of polynuclears has its normal variation, depending on the age, being lower in children. Polynuclear counts ran as high as 94 per cent (Case 26). A high white cell count with moderately increased percentage of polynuclears, I believe to be a more desirable condition than the reverse. In the early stage of the infection when the sepsis is severe the eosinophiles disappear from the blood count. As the patient improves, with lessening of the sepsis, they return. A reappearance of eosinophiles is a sign of improvement. In our laboratory we are accustomed to count all the young forms of polynuclears as one group. Generally when this group is more than 15 per cent it indicates that the sepsis is becoming severe enough to cause noticeable irritation or disturbance of the bone marrow. In the very severe degrees of sepsis the percentage runs as high as 40 per cent or more. In one case (No. 31) the young form percentage went to 63 per cent, with a total white count of 36,050, and a polynuclear percentage of 90.5 per cent. There were no myelocytes reported. It is the highest young form count I have seen in one who recovered. When the young forms appear as high as 40 to 50 per cent, or more, one may expect to see an occasional myelocyte. The appearance of myelocytes is of course always of grave significance. Sometimes the total white count will drop from a previously high mark to normal or slightly less with no improvement of the patient clinically. In such instances the polynuclear percentage and the young form percentage can be very illuminating. In one case (No. 50) the total white count was 7200, polys 50 per cent, eosinophiles 0 per cent, young forms 35 per cent, with a fever at the same time of 105.4 degrees. No myelocytes reported. The patient died. In another case (No. 44) a count was made six days before admission to the hospital, and was as follows: Total white count 23,900, polys 64 per cent, young

forms 30 per cent. On the day of admission to the hospital total whites 6,500, polys 35 per cent, young forms 57 per cent, with one basophilic myelocyte. In 48 hours the total white count was again 14,800, polys 44 per cent, young forms 53 per cent. This child received extensive drainage and recovered. In all severe neck infections which I have seen there has been a well marked secondary anemia. I have until now not seen a marked leucopenia in a deep neck infection. Considering that agranulocytosis frequently begins with mouth and throat inflammation, it is possible that a deep neck infection might occur. What the blood picture would be in such a case, I do not know.

PHARYNGOMAXILLARY SPACE INFECTION (PARAPHARYNGEAL)

Infection of the pharyngomaxillary space is most often caused by infection from the tonsils and pharynx. As soon as infection has penetrated the tonsil fossa muscle bed it has become pharyngomaxillary, and gives rise to symptoms and signs which characterize infection of this region. Occasionally, however, it does not localize in this area with the formation of a large abscess, but forms a small abscess which gravitates and extends downward along the buccopharyngeal fascia, becoming a pretracheal fascia infection. There may be multiple minute abscesses. In such instances the tissues along this fascia become greatly thickened, and there may or may not be abscess formation along it.

The local symptoms and signs indicating a pharyngomaxillary space infection differ according to whether the infection is situated in the anterior or posterior part of the space; that is, whether in the prestyloid or retrostyloid part. Both parts may be involved.

Batson[22] has with good reason stressed the conception that the neck is composed of tissue masses (bones, muscles, viscera, vessels, etc.) held together by connective tissues or fascias. He states that these fascias are capsular, areolar and very dense perivascular. The perivascular fascias with the vessels form pedicle-like structures connected with the neck viscera.

The carotid sheath and its contents form the largest of these pedicle-like structures. It is located in the posterior or retrostyloid compartment of the pharyngomaxillary space, and, I believe, is of greater influence in the determination of the location and behavior of an abscess in this region than are the structures attached to the styloid process. An abscess located posterior to this vascular pedicle can be approached from behind the sheath, keeping very close to

the scaleni muscles in going inward. Sometimes it may be reached by incision of the lateral pharyngeal wall through the mouth, well behind the posterior pillar. It may also sometimes be reached through the posterior wall of the tonsil fossa after removal of the tonsil. It may also be drained by exposing the great vessels below the posterior belly of the digastric muscle, and proceeding upward along the sheath to and beneath the styloid process. The vascular pedicle is very dense, and unless partly disintegrated or softened by the inflammatory process, it is not possible to penetrate it with the finger without inflicting dangerous trauma on its contained structures. If the infection is anterior to the styloid and its attached structures, there is swelling of the lateral pharyngeal wall, displacement of the tonsil and fauces toward the median line, and trismus. Tilting of the uvula accompanies this in many cases. There may also be swelling of the parotid gland region. There is, first of all, painful and difficult swallowing. It should be differentiated from peritonsillar abscess, and the two may be present at the same time. Where there is acute peritonsillar abscess there is also inflammatory swelling of the tonsil itself, whereas with pharyngomaxillary infection the tonsil may be, and most frequently is, normal in size. There may be no inflammatory redness of the pharynx and fauces. With these local manifestations there is sepsis which may be mild or very severe and alarming. Given a case with well marked local manifestations, it is extremely important to anticipate the sepsis and recognize it at the earliest possible moment, so that appropriate and adequate drainage may be applied before disastrous blood stream infection has occurred. Swelling of the submaxillary and lateral regions of the neck is usually of moderate degree, and is caused mostly by inflamed and swollen lymph glands. If drainage of the pharyngomaxillary space is carried out the inflammation in the glands subsides. It is possible for a cervical gland abscess to form in these cases, but not common.

If the parapharyngeal infection is deep to the styloid process and its attached structures, the local symptoms and signs are different, in that there will be no trismus and usually no swelling of the parotid region. The trismus is absent because the infection is not near enough to the internal pterygoid muscle to cause spasm, or splinting. There is, however, swelling of the lateral pharyngeal wall, and usually swelling of the posterior pillar. There may be or may not be a slight prolapse, or displacement inward, of the tonsil. There may be some swelling of the parotid gland region, but it is more often absent. A very direct approach to such abscesses may be made through the posterior pillar after removing the tonsil.

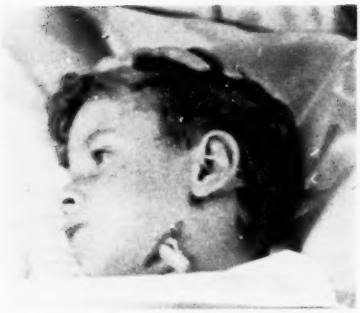
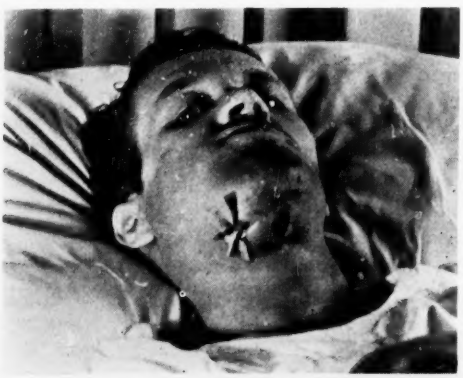


Fig. 1. Case No. 76. Drain in cervical abscess and on sheath. Abscess situated between sheath and sternomastoid muscle. Tonsils removed one month after neck healing was complete.



Figs. 2 and 2a. Case No. 67. Submaxillary infection (Ludwig's Angina) following dental infection and extraction. Abscess drained through midline incision. Later osteomyelitis of mandible developed. Abscess drained through lateral incision, and subsequently sequestra, as shown in Fig. 2a removed.

This blunt thrust may then be made with the instrument in the sagittal body plane. In those cases having only a mild sepsis this is a very justifiable procedure, and will frequently avoid external neck drainage. With severe sepsis external drainage should be done. If the severe sepsis should occur after internal drainage with or without tonsillectomy, external drainage is indicated. Painful and difficult swallowing is of course also present in this type. In all cases of parapharyngeal infection which I have observed there has been present injection of the homolateral ear drum. This may be very slight, but can be recognized. It appears on Shrapnell's membrane, the margins of the drum and the malleus handle. The infection is in close proximity to the auditory tube, and it would be surprising indeed not to get some tympanic evidence of it. There may be otalgia.

The foregoing symptoms and signs indicate uncomplicated pharyngomaxillary, or parapharyngeal, infection. If not drained during this stage, infection of the internal jugular vein is a strong possibility. It is then regarded as extension to the carotid sheath compartment and constitutes a complication. The earlier the neck is drained after well marked sepsis is present the fewer complications will be observed. This has been by experience, and accounts for the comparative scarcity of complications in my later series of cases.

Not infrequently one sees an abortive type of pharyngomaxillary infection, particularly those in which the infecting organism is the hemolytic streptococcus for which sulfanilamide has been given. When such spontaneous improvement progresses uninterruptedly, surgery may be withheld. All local and general signs of infection should have disappeared before one is justified in relinquishing close watchfulness.

CAROTID SHEATH INFECTION

Doctor Mosher has called this the "Lincoln Highway of the Neck," and it is in relation with all fascial planes (Mosher[3]). Infection along this sheath is always secondary to infection along one or more of the other layers of the cervical fascia, or secondary to contacting infected cervical lymph glands. Most frequently it follows infection in the pharyngomaxillary space. Infection in the submaxillary region, of nondental origin, is very likely to be a precursor. The vast majority of pharyngomaxillary and carotid sheath infections is caused by infection in and about the tonsil (Mosher[7]). Frankel[8] believes the pathological sequence is caused

by thrombosis of the peritonsillar veins; Uffenorde[9] that the pathway is through the lymphatics; Waldapfel[10] that there is perivascular extension directly from single or multiple abscesses in the peritonsillar region. A combination of these pathways of infection probably embraces the truth. I believe that the more rapidly and the more completely the inflammation localizes and forms a solitary abscess, the better the prognosis, providing that it is at such a time reached by drainage. Thrombotic advances of the inflammation soon reach the blood stream with unpredictable and unpreventable involvements. The more predominantly it is cellulitic in character, the more difficult it is to drain, and the more grave the prognosis.

There are no characterizing local symptoms and signs of infection of the sheath or its contents. Such symptoms and signs as do occur indicate that there is inflammation beneath the sternomastoid muscle, most frequently inflamed glands. There is a diffuse swelling along the muscle, and the individual glands cannot be palpated. There may be a tender spot just below the angle of the jaw. If there is swelling along the muscle there is marked tenderness, and usually torticollis toward the opposite side. Induced contraction of that sternomastoid muscle by placing resistance against the opposite side of the chin with the hand is painful. One should not expect to be able to palpate the cord-like feeling of a thrombosed vein in neck infections because of the presence of inflamed glands and swelling of the parts. If there is abscess formation in the inflamed glands there may not be fluctuation, because the glands are deeply situated. Frequently, however, there is edematous pitting on deep pressure with the finger. If this sign is obtainable it is fairly good evidence that there is underlying pus. Abscess formation is not so common in the deep chain of lymphatics as in the superficial chain, but often follows abscess in the superficial chain. Usually the glands are the seat of well-marked inflammatory swelling, and are quite adherent to the sheath which often shows inflammatory thickening. In this event it will be necessary to carefully excise some of them in order to expose the vein if ligation or resection is performed. It is a tedious procedure. Even where the vessel is not ligated, free exposure brings about better drainage, and tends to prevent thrombosis. Subsequent sloughing of lymph nodes with possible secondary hemorrhage from their vessels of supply is thus avoided. Secondary hemorrhage from small veins occurred in one of my cases where the glands were not excised at operation (Case 37). In another case (Case 63), where the sheath was inflamed and thickened, it was necessary to pinch off a small bleeding vessel in it. No free pus present.

Evidence of blood stream infection is entirely general in character, and is indicated by sepsis. There are four findings which, when present at the same time, definitely indicate a blood stream infection, viz., septic blood count picture, abrupt rises and drops in temperature, chills and sweats, and positive blood culture. Of these the last is the least constant. Chills are not so often present in children as in adults, though I have seen severe chills in a child four years of age (Case 29). Sweats, day or night, are often present without chills in both children and adults. The temperature curve is characteristic. The blood count is variable, but not so variable if the young forms are counted. By this is meant that while a high total white count and an increased polynuclear percentage are expected, sometimes these phases of the count are not much over normal, and one may be somewhat puzzled. If the young form polynuclears are counted, often the percentage of these will be found to be greatly increased. I have come to regard this as evidence of a septic blood picture. It is particularly helpful in the infections in other parts of the cervical fascia.

Chills are inconstant, almost as much so as the positive blood culture. This is especially true in children. When present, a chill or two is positive evidence. When absent, other symptoms and signs must decide the issue. Occasionally chilly sensations can at least be suggestive. Sweats, whether associated with chills or whether occurring without chills are significant, and in the latter instance are common. When sweats are profuse and frequent, dehydration and loss of salt are increased, and rational therapy demands that such losses be corrected by sufficient administration. Chills do not always indicate actual thrombosis in the vein. It is well known that a severe acute tonsillitis may be ushered in by a chill. I have observed a chill occurring in the beginning of an acute pharyngitis and tonsillitis before the patient was aware of the existence of a severe inflammation in the throat. Frequently these go on to a neck infection immediately, but frequently also, they make an apparent recovery, and then after from one to six weeks have a sudden recurrence of sore throat, and go on to a neck infection. My histories indicate that pretracheal fascia infections often begin in this manner. Chills of this character are, I believe, caused by a shower of toxic infectious material suddenly released into the small radicals of the venous blood system, and then quickly eliminated. It may be likened to an April shower. Where chills occur at a later period of the neck infection, from a continuous toxemia, a definite blood stream infection may be assumed. This may be likened to a prolonged storm. This phe-

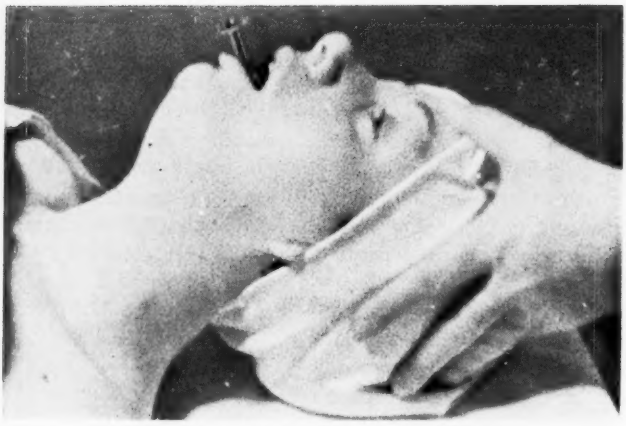


Fig. 3.

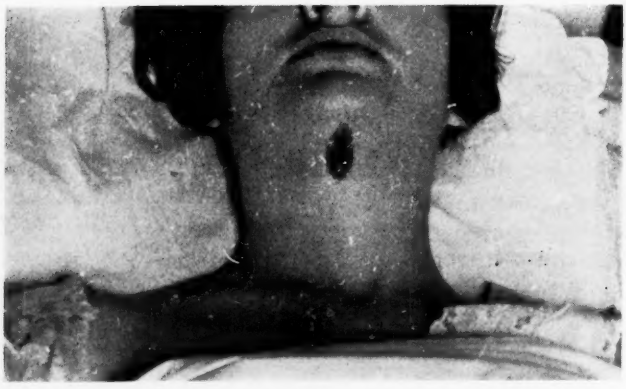


Fig. 3a.

Figs. 3 and 3a. Case No. 45. Submaxillary infection (Ludwig's Angina) secondary to infection of lingual tonsil. Midline incision exposing fairly large abscess cavity. Fig. 3. On operating table before operation, showing breathing tube in position.

nomenon of chills is one of the manifestations of the activity of the clearing mechanism in the presence of septicemia (Kolmer[25]). A constant invasion of the blood stream is believed to occur, resulting in sufficient accumulation of bacteria and toxins to produce reaction prior to their elimination. It is not believed probable that bacteria undergo much, if any, proliferation in the blood.

Because of this dependence on the general manifestations for diagnosis, it becomes necessary to exclude other conditions which cause sepsis, particularly those that give the typical temperature curve. Prominent in this respect is pyelitis, which is of particular importance in female children. Mastoiditis is another possible cause of blood stream infection, and may be actually co-existent (Case No. 16). Empyema likewise. There are many others.

Definite evidence of existing or impending blood stream infection is the sudden appearance of a distant inflammation. Such manifestations are somewhat variable. A sudden, painful tender inflammatory swelling of the dorsum of the foot, calf of the leg (Cases 24 and 27), of a joint, or some other subcutaneous area is strong evidence of existing or impending blood stream involvement. Deeper regions, such as a long bone, a hip joint, the pelvic tissues, the lungs, or the mesenteric tissues may be thus involved. If such secondary lesions go on to abscess formation, it is necessary that they be drained surgically. If they occur early, before any neck drainage has been done, immediate drainage of the neck may result in spontaneous subsidence of the secondary lesion (Case No. 27). If neck drainage is delayed or if thrombosis of the internal jugular vein occurs after the neck has been drained, in other words, if the distant complication is a late occurrence, it is probable that it will be necessary to drain it, especially if it is a joint that is involved. It is no doubt possible that pulmonary and cerebral abscesses may be thus caused, but until now I have not observed any.

When infection of the blood stream and jugular thrombosis are suspected or known to exist, the eye fundi should be frequently examined for chocked disc and retinal thrombosis. After cavernous sinus thrombosis has actually taken place the outlook is hopeless. Choked discs do not always signify cavernous sinus thrombosis, but they do indicate serious embarrassment of intracranial circulation. Unless it is very transient, serious loss of vision from optic nerve atrophy will result (Case No. 24). Actual thrombosis of the internal jugular vein is not always present even when the vein walls are involved (Case No. 12). The most important consideration is to get drainage as near as possible to the portal of entrance, or

into the pharyngomaxillary space adjacent to the tonsil. Next it is important to place drains on the widely exposed carotid sheath. If thrombosis can be immediately demonstrated, ligation or resection is immediately indicated. If not it is probably safer to do so anyhow if the sepsis is severe. The important thing is to drain the original abscess or site of infection. When blood stream infection is imminent it is very important to start intensive medication, and the use of specific, convalescent or polyvalent sera given early can be of great value. Bacteriophage is particularly indicated for staphylococcic infections. Transfusion repeated frequently is very valuable. Immunized donors are to be preferred, particularly if they are members of the patient's family. One may sometimes improve a donor with typhoid immunization. Transfusions may be given every two or three days. The sulfanilamide preparations are indicated for streptococcus hemolyticus infection. For dehydration 3000 cc. of 5 per cent glucose in saline to an adult per day. Sera are more valuable as prophylactics than as curatives, therefore should be used early.

It is not considered necessary to do more than mention general measures of therapy in neck infections. The application of heat directly to the inflamed parts I consider valuable and beneficial, for which reason irrigation of the mouth and throat with hot saline solution with a bag temperature of 115 to 120 degrees F. is routinely carried out at two-hour intervals. If too exhausting for a given patient the frequency may have to be diminished.

Externally heat or cold may be preferred. Sometimes it is well to be guided by the preference of the patient. In our practice if the ice bag is applied externally it is in conjunction with hot saline throat irrigations. With medical diathermy I have had no experience. Irradiation with the roentgen-ray was used in some of our cases, but cannot be definitely evaluated, because drainage was done for the usual indications. To what extent the ray assisted in the recovery it is therefore not possible to state. In one case (Case No. 31) in which erysipelas developed after recovery from the neck infection, it was most effective. It is my impression that it is valuable particularly in those cases characterized by a hard, brawny cellulitis with or without dermatitis, such as accompanies Ludwig's angina, also where the superficial tissues are involved.

Where there is inflammatory edema of the larynx causing hoarseness and dyspnea, steaming inhalations from the croup kettle are beneficial, and when dyspnea becomes alarming the oxygen tent with or without helium is a life saving measure until the air-way

is completely restored by surgical procedure. Other measures are mentioned elsewhere in this paper.

SUBMAXILLARY SPACE INFECTION

By this is meant those in which the infection first involves the region below the mandible in the submaxillary and submental triangles, parotid region and the upper part of the cervical region adjacent to these triangles. Ludwig's angina falls into this group. It frequently happens that it is in these regions that the first manifestations of a deep neck infection make their appearance. There are two types, one dental in origin and the other not.

INFECTION OF DENTAL ORIGIN

This follows dental caries, pyorrhea alveolaris or alveolar abscess. It more frequently follows extractions, particularly when local anesthesia has been employed. It would seem that it is inadvisable to inject a local anesthesia into or near an inflamed gum or even into one which is likely to become inflamed secondarily to the dental or alveolar infection. There is frequent evidence that such a procedure has preceded the neck infection. A distant nerve block is probably less risky. A general anesthesia or postponement of any radical dental operation would seem the better policy. It is of course conceivable that infection may follow an extraction performed when no apparent infection is present. I do not believe anything more definite than this can be stated, except to suggest that the gums be very carefully examined before proceeding, including bacteriological studies. Infection of the gingival tissues, floor of the mouth and neck may result from dental caries involving the crown, and not necessarily the roots of a tooth. This particularly in the case of a lower third molar. There is often a high fold of gingival tissue just posterior to this tooth, making a deep pocket or recess between it and the tooth crown, in which detritis may accumulate and be the starting point of an infection. This was the sequence of events in one of my cases (No. 35). In the majority of instances infection from the teeth extends downward along the lingual side of the mandible, in which case the floor of the mouth is quickly involved. It is accompanied by trismus. The infection may, however, extend downward along the buccal side of the mandible, and involve the face and neck, but in this event the floor of the mouth is not involved, and there will be only slight trismus caused by embarrassment (pain and spasm) of the masseter and buccinator muscles. At the beginning, therefore, marked tris-

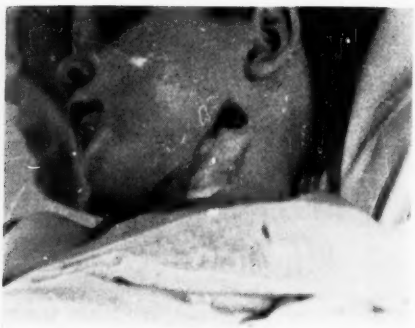


Fig. 4. Case 37. Carotid sheath infection with blood stream involvement. Also pneumonia followed by empyema requiring thoracotomy. Full exposure of carotid sheath. Secondary hemorrhage from sloughing lymph glands which were not excised at the primary drainage operation. Illustration shows drain in pharyngomaxillary fossa.

mus would be absent. The infection may travel along both sides of the process. Where there is frank alveolar suppuration at the time of the cervical infection, removal of the affected teeth should be considered at the same time that the neck is drained. A radiograph can be of considerable assistance.

The applied anatomy of this region is interesting and important. The superficial layer of the deep fascia in this region finds attachment above, to the lower border of the jaw in front of the neck. Laterally it covers the masseter, encapsulates the submaxillary and parotid salivary glands, and above, takes attachment on the zygoma. From these points it extends downward, and is firmly attached to the hyoid bone and thyroid cartilage. The lymphatics of the tongue, floor of the mouth, and teeth of the mandible course downward toward the hyoid bone, above which they turn laterally and empty into the median upper deep cervical glands on the carotid sheath at about the level of the larynx, and, on the way, some of them drain into the nodes in relation with the submaxillary salivary gland. These, then, are the glands into which infection in

these parts eventually drains. I have in the past observed early internal jugular vein thrombosis in these cases of submaxillary infection when drainage is delayed (Case 24). The lateral course of the infection after it reaches the neck can therefore be explained on these two points, viz., the fascial attachments and the lymph drainage route. The mouth is separated from the neck by the mylohyoid muscles. Anteriorly and laterally this muscle sling is attached to the inner surface of the mandible. Posteriorly its median portion is attached to the hyoid bone. This muscle sling with its enveloping fascia is therefore part of a relative barrier to infection of the neck from infection of the teeth and floor of the mouth above it. Infection above it, in addition to penetrating it through the lymph channels and tissue spaces, passes backward and laterally around the free part of its posterior border into the neck. Along this route there will be involvement of the submaxillary salivary gland, which winds itself around its posterior border. The submaxillary salivary gland is sometimes excised in draining such infections (Ramsdell[11]). Thomas[2] believed that the buccal or intraoral involvement in Ludwig's angina was secondary to the neck involvement by this pathway. He demonstrated an actual muscular opening in which lies the posterior or lateral part of the submaxillary salivary gland. Suppurative inflammation sometimes primarily involves the submaxillary salivary gland as a result of the presence of an obstructing calculus, either in the gland itself or in its duct. In such cases both the mouth floor and the neck, i. e., above and below the mylohyoid muscle sling, are simultaneously involved. If the calculus is in the duct and can be removed, drainage through the duct or through the mouth floor will often be effective. If the calculus is in the gland the best remedy will be drainage or excision of the gland.

A description of the symptoms and signs of submaxillary region infection of dental origin applies exactly to a description of Ludwig's angina, though the latter is not always caused by dental infection. Most frequently submaxillary infection occurs after a dental extraction, and its progress is usually rapid. Following the extraction there is persistent pain, tenderness and swelling in the area for from one to five days. If the infection travels on the lingual side of the alveolar process, there is immediate swelling of the floor of the mouth. This increases and spreads to the tongue, which is at the same time crowded upward in contact with the palate. There is usually a foul breath odor and marked trismus. With involvement of the mouth floor and tongue there is elevation of temperature,

after which a definite sepsis is established. Often all this happens in just a few hours. Pressure over the submental and submaxillary triangles causes marked pain in the mouth floor even before there is marked swelling of these neck regions. This neck swelling follows very soon after involvement of the mouth floor, and is very tense and boardy to the touch. Frequently the temperature rises to unusual heights. It is variable, and I have seen it as high as 106 degrees (Case 22). If high it is very apt to be accompanied by chills. The patient is very miserable. The pain is severe. Chewing and swallowing are very difficult, painful or impossible. Breathing is somewhat difficult. The sepsis is often very severe, and the toxemia profound. There may be sweats. At this stage it is difficult or impossible to examine the deeper parts of the mouth and pharynx. No individual lymph glands can be palpated because of the swollen and indurated neck region. It is a most pronounced cellulitis, and the whole anterior and lateral neck region may be involved. As a rule, one has decided to drain before the swelling has reached below the level of the larynx. If the infection penetrates the tongue or begins at the base of the tongue, edema of the larynx is a strong possibility; and if the infection is not checked a tracheotomy may be necessary. If seen early, before there is marked neck swelling, it may be possible to locate an abscess, and drain it through the mucosa of the mouth floor. This may bring about subsidence of the infection. Houser[14] has reported a number of such cases. If, however, no abscess is located by this procedure, external drainage is immediately indicated. Sometimes an abscess is not immediately found by the external route, but frequently profuse drainage occurs without further surgery after one or two days. If the infection involves chiefly the submental region, incision in the median line of the submental triangle is to me the incision of choice. Through this approach no large blood vessels nor any other structures of importance are encountered, and the incision may be carried as deeply as necessary to reach between any of the muscle planes. Through it one may explore laterally to either side on any level. Irradiation is sometimes helpful in this type of case, but does not supplant drainage (Galloway[27]).

When the infection is chiefly unilateral, an incision close to and paralleling the margin of the jaw may be found the most direct approach. Through this the submaxillary salivary gland can be freely exposed, and access to the posterior part of the mouth floor obtained where it winds around the posterior border of the mylohyoid muscle. The deep fascia attached to the border of the jaw

may be penetrated bluntly, and access afforded to the lingual side of the alveolar region where abscesses frequently localize. When an alveolar infection travels downward along the buccal side of the jaw, there is no need to penetrate the fascia attached to the jaw margin.

Occasionally, in addition to the involvement of the neck, there occurs an osteomyelitis of the mandible. If, after reasonable time after neck drainage has been established, between one and two weeks, infection in the region of the mandible has not shown evidence of improvement, osteomyelitis should be suspected. In the two cases in this series (Cases Nos. 30 and 67) the first radiographic evidence of osteomyelitis appeared at the end of the second week, this evidence being reported as "osteoporosis." Definite evidence of sequestrum formation was not present until two weeks later. X-ray evidence of osteomyelitis in Hochfilzer's case[12] is corroboratory of this. These sequestra must of course be removed as they form and drainage maintained throughout. Food requiring mastication must be avoided, since there is the possibility of pathological fracture. When the last sequestrum has been removed healing with recovery is quite rapid. It has been our custom to remove all teeth situated in the involved area. Carmody's[28] recent article on this subject should be read.

Otalgia, homolateral, may be present early, but there is no injection of the drum unless the infection gets into relationship with the auditory tube. If the tongue is crowded backward and upward, and particularly if it is at the same time swollen, a lateral neck radiograph will disclose the tongue base and the epiglottis contacting each other. In other words, the fairly wide normal space between the tongue base and the epiglottis, the valleculæ, as shown in a soft tissue exposure, will be obliterated.

In the typical case of Ludwig's angina there is not only marked pain on swallowing but also marked pain when chewing is attempted. The reason is obvious. In severe cases chewing is impossible. In the literature the most frequent cause of Ludwig's angina has been reported as infection from the teeth (Blassingame,[13] Houser,[14] Thomas,[2] Ashhurst[15]). One very typical case in this series began in an infection at the base of the tongue in one lateral half of the lingual tonsil (Case No. 45). There was one other case (No. 52) in which the infection followed extraction of a lower second molar tooth, and localized in the pharyngomaxillary space where an abscess was drained. The infection was introduced by the needle in

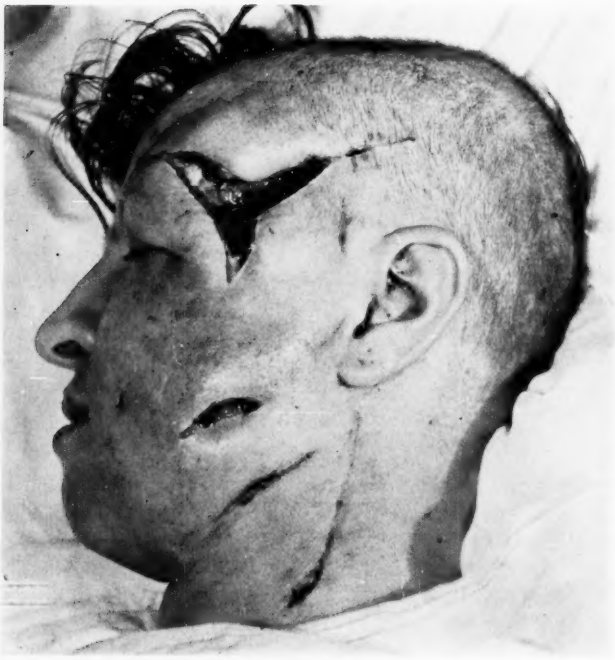


Fig. 5. Case 68. Zygomatic and temporal fossa infection resulting from dental infection following extraction of upper third molar tooth under local anesthesia. Necrosis of maxilla with sequestration. Pharyngo-maxillary infection without abscess formation. Preliminary ligation of external carotid artery to prevent hemorrhage from extensively sloughed soft parts above in temporal fossa.

making the mandibular nerve block, and invaded tissues deeply on both sides of the ramus of the jaw, including the pharyngomaxillary space. At no time was there involvement of the floor of the mouth, but there was marked swelling of the cheek as high up as the zygoma and extending downward across the submaxillary and upper cervical regions. The pathway was on the buccal side of the alveolar process from the dental disease, and to the pharyngomaxillary space from the needle. The prognosis in these dental cases is good if early drainage is established. If not, edema of the larynx, pneumonia and blood stream infection are possibilities.

SUBMAXILLARY INFECTION NOT OF DENTAL ORIGIN

Some of the severest cases of neck infection fall into this group. Often the antecedent sore throat is comparatively mild in degree, and of short duration. For example, a patient may retire to bed in the evening with a mild sore throat which during the night increases rapidly in severity, and by morning the patient already exhibits submaxillary swelling. At the same time there is fever. There is usually no trismus, nor is it characterized by foul breath odor. In from one to three or four days there may occur a sharp chill. The submaxillary swelling is usually quite indurated and diffuse. There are usually no palpable lymph node swellings, and sepsis is pronounced. I have previously stated that these are dangerous and insidious cases, and are very apt to bring about the severest complications. A tonsillitis, a pharyngitis, infected tonsil remnant, a sinusitis are, one or more of them, the most common starting points. Drainage is usually necessary before an abscess has had time to form. This type of case has a decided tendency to extend to other fascial planes, which is why it is a dangerous type. It most often extends laterally to the vessel sheath with resultant invasion of the blood stream (Case 24). It may also extend to the pharyngomaxillary space. The pretracheal fascia is also occasionally involved. The deep fascia is firmly attached to the hyoid bone, which, together with the direction of lymph drainage, tends to favor the spread of the infection to the side of the neck. The swelling of the neck involves the submental, submaxillary, parotid and adjacent upper cervical regions, usually unilateral. Drainage aims at the sheath compartment, and if the blood stream is involved as evidenced by chills, temperature curve and positive culture, ligation of the vein is indicated. Usually no free pus is encountered, even when drainage is delayed, but in such an instance I have seen the vein completely necrosed for a considerable distance, and replaced by a thrombus (Case No. 24).

As is to be expected, sepsis is very severe. In such cases it is advisable also to place a drain in the pharyngomaxillary space. With the extension of the infection from the first region involved, there may take place a lessening of the visible neck swelling in the submaxillary region. One must be guided more by the degree of sepsis than by local signs. Diminution of the neck swelling is of itself no evidence of improvement.

To be called "Ludwig's Angina" the process must show certain definite characteristics. There is, somewhere in the locality of

the base of the tongue, floor of the mouth or alveolar process of the mandible, an abscess which it may not be possible at first to find. At the same time there is a marked cellulitis involving the mouth floor and tongue which extends into and involves the submental and submaxillary regions of the neck. Almost all of this swelling is caused by intense cellulitis, and has almost a bony hardness. Trismus is marked. There is foul breath odor. There may be embarrassed breathing from the swollen and crowded tongue. The most frequent complication is edema of the larynx. The term "Ludwig's Angina" is not used where the characteristic cellulitis is absent.

PRETRACHEAL FASCIA INFECTION
(Buccopharyngeal and Visceral)

The applied anatomy demonstrates this as the enveloping layer of the viscera of the neck. For the sake of brevity, when the term "pretracheal" is used in this paper, the pretracheal, the buccopharyngeal and the thyroid gland parts of the visceral fascia are designated. The part of the buccopharyngeal fascia referred to here is all that which lies below the superior constrictor muscle.

Pretracheal fascia infection may be primary or secondary. What is meant in this regard is that the tissues along this layer may be the first to be involved when the inflammation extends into the neck; or this layer may be involved secondarily by extension of the infection from other regions or fascial planes as, for example, extension from the prevertebral fascia or the pharyngomaxillary fossa. Trauma from food, foreign bodies or instrumentation are examples of primary involvement. Late extension from a focus of pus outside the tonsil is an example of secondary involvement, as it is at first a pharyngomaxillary, or parapharyngeal, infection.

Infections involving the pretracheal fascia are always very serious. They are characterized by severe dyspnea approximating asphyxia, even when there is no swelling nor edema of the mucosa of the larynx or trachea. In one such case (Case No. 50) on whom autopsy was performed, there was found macroscopically, extensive bronchopneumonia with scattered areas of consolidation, and microscopically, extensive bronchiolitis. The dyspnea was out of proportion to any demonstrable respiratory obstruction, ante or postmortem. Prognosis in cases of pretracheal fascia infection should always be very guarded. Treatment is often futile, because in so many of the cases the involvement of this plane is a late manifestation. A tracheotomy is often performed as a desperate effort to

prolong life. An earlier drainage of the fascial plane is much more apt to be effective. When the search for pus is delayed until the time when a tracheotomy is necessary, the prognosis is indeed bad. If pus is present in such cases it is very difficult to keep it out of the trachea. A pyogenic pneumonia is a strong possibility, and usually also involvement of the mediastinum. I remember one case in which a tracheotomy became necessary 24 hours after surgical drainage of the neck was performed. When seen in consultation 48 hours later, there was more pus being coughed out of the tracheotomy tube than was draining from the fascial compartment. Chest radiograph demonstrated pneumonia and mediastinitis. The nearer the drainage can be applied to the starting point of the neck infection the better the prognosis, particularly if it is not done late. Several regions widely separated may require simultaneous drainage.

A knowledge of the cause and the portal of entrance of the infection is of great assistance in the placing of drainage. A few minutes spent in getting this part of the history are well repaid. The primary focus may be very small, in which event there might be few objective signs to help in its detection, and one is compelled to rely almost entirely on subjective symptoms. As an example of this, the primary focus outside a tonsil may be so small as to be entirely overlooked in an autopsy specimen unless a deliberate search is made for it. When infections enter through the esophageal wall as a result of trauma, the region is deeply invaded before any defensive reaction occurs in the parts, and chest extension with its extreme sepsis results before there is any recognizable evidence of its presence in the neck. These are the severest of all varieties of neck infection, and are practically hopeless from the start. Occasionally there is localization with abscess formation at the site of injury (Orton[16] and Case No. 4), in which event the prognosis should be much better. Infections of this kind were formerly frequent after operations on the esophagus, but the mortality has been almost eliminated by the performance of the two-stage operation. Preliminary drainage of the fascial plane is accomplished in the partial or complete skeletonization done as the first stage of such operations. Large quantities of pus have been evacuated by drainage along the pretracheal fascia with slight or no visible preexisting swelling of the neck. Indeed, it has happened that the purpose of such an operation was tracheotomy for the relief of dyspnea, and that the presence of pus had not been suspected. In Richard's[17] case the absence of neck swelling can be attributed to the large space afforded the adjacent reservoir of pus in the upper

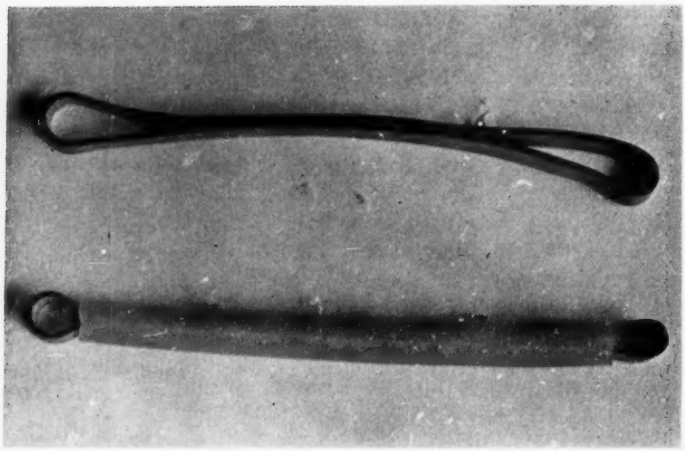


Fig. 6. A type of soft rubber drainage material devised to avoid extrusion from wounds in parts where movement cannot be entirely eliminated. Does not require suturing nor clipping to wound margins. Is composed of an ordinary rubber band over which is cuffed a piece of Penrose tubing of suitable size as shown in illustration. The free loop at the end allows soft tissues to fall into its confines, thus tending to hold it fixed. It has been quite satisfactory where extrusion of drains must be avoided, and where, otherwise, some type of gauze packing would be necessary. The shorter the cuff, the larger the size or diameter of the loop.

part of the chest. It is also possible that the neck infection may be secondary to the chest abscess, whether this be mediastinal or pleural. A fistula sometimes extends from a focus of pus outside the tonsil, downward into the neck along the pretracheal fascia to a point lateral to the larynx or trachea (Richards, G. L.[18] and Case 51). Roberts[19] reports draining a perilaryngeal abscess which had extended into the mediastinum, through the homolateral pyriform sinus.

In this series of cases there are eight, with two deaths (Cases 31, 40, 44, 46, 50, 51, 4, 21). The two that died were seen late, one dying eight hours after arrival at the hospital. One followed an attack of tonsillitis and pharyngitis, and the other tonsillitis and quinsy. Of those that recovered, one (Case No. 4), followed in-

jury by a bread crust, and was drained by internal incision through the laryngeal speculum. Another (Case No. 21), was attributed to infection of a lymph node in relation with the thyroid gland fascia. The remaining four all followed pharyngitis, two of them also having had tonsillitis and bilateral cervical adenitis involving both superficial and deep chains. One of these two had abscess of the glands. Where such an onset history is obtained, and during the course of the illness the patient develops dyspnea, drainage should be placed not only along the pretracheal fascia, but also into the pharyngomaxillary space close to the tonsil on the involved side.

The distinctive symptoms and signs of pretracheal fascia infection arise from the viscera involved. Infection lateral to the middle and inferior constrictors of the pharynx immediately causes painful and difficult swallowing. Swelling and redness of the wall of the hypopharynx, usually unilateral, should be looked for. The first complaint, when infection originates in the pharynx or tonsils, is sore throat. All these symptoms may at first be mild. The first indication that the pretracheal fascia is affected is hoarseness. In infants and children this symptom is not always noticed by the parent, and is soon followed by some degree of dyspnea. In adults dyspnea is apt to be a later symptom. Sometimes there is no actual hoarseness, but instead there is a gutteral or muffled voice indicating that the laryngeal swelling or edema is higher than the vocal cords. A muffled voice may be present with swelling limited to the hypopharynx. It may be possible to elicit tenderness on pressure over the laryngeal and hyoid regions laterally (Orton[26]). There is at first no visible swelling of the neck. In children there may be pre-existing inflamed and swollen glands on one or both sides. One of my adult cases (No. 46) developed infected glands on the homolateral side in which an abscess was drained subsequently. Later there occurs some swelling of the neck which in this type of case is apt to be moderate. At first it is indurated and diffuse. If localization occurs there may or may not be fluctuation. Occasionally, where fluctuation is not elicited, there may be present a pitting on deep pressure with the finger. When present this is a very important sign, for in my experience it has never failed to indicate underlying pus. It is my belief that it is more frequently to be elicited than fluctuation.

Mirror examination of the larynx and hypopharynx, when possible of performance, reveals involvement of these structures. There may be swelling of the lateral wall of the hypopharynx, the pyriform sinus and also of the glosso-epiglottic fold; also inflam-

matory swelling and edema of the epiglottis which at first is limited to one lateral half. If the process extends the swelling then advances to the aryepiglottic fold and the arytenoid region. Still later the rest of the vestibule and the ventricular band become edematous, and it is no longer possible to see the vocal cord on that side. Further involvement of the larynx causes dyspnea from involvement of the whole of the laryngeal interior. While this involvement of the larynx is taking place, the infection in the neck is making a corresponding advance, and the question of drainage must be given consideration. Body temperature is apt to be continuously between 101 and 103 degrees. In my experience the temperature does not run as high as in the submaxillary region cases, and chills are not so frequent.

At this stage swallowing is very difficult, and there is some danger of fluids getting into the air passages. This condition is serious, for the patient ceases to make the necessary effort, and dehydration is the result. The fluid intake must be very carefully recorded, and if the patient will not or cannot take enough by mouth it must be given subcutaneously or by vein. Sometimes a feeding tube may be employed in order to minimize the use of needles through the skin. If the infection involves the esophagus, the tube should be left in place and all food and drink given through it. If the involvement is chiefly laryngeal and tracheal the patient may much prefer to swallow and accept the accompanying pain. A tube of small caliber is less likely to cause ulceration than a larger one. If there be regurgitation of fluid through the nose when swallowing is attempted, the use of the tube may become obligatory. Dehydration must receive daily consideration, and, if the blood condition indicates it, fluid administration must be supplemented by transfusion every three to five days in severe cases. Specific medications such as serums, bacteriophages, anti-virus filtrates and chemical agents (sulphanilamides) can be very beneficial when intelligently employed. Transfusions from immunized donors are also valuable. Hot saline throat irrigations are very valuable.

Occasionally infection of the pretracheal fascia is caused by infection from the thyroid gland or its fascia (Case 21). There is a rich distribution of lymphatics at this location, and there is a rich blood supply. The chief difficulty in such cases is differential diagnosis which, in the absence of sepsis, is not always easy until there is definite fluctuation. If the infection is severe and accompanied by marked sepsis it may not be advisable to wait for fluctuation or pitting. Acute thryoiditis should be ruled out. It should

be borne in mind, however, that the infection may be limited to its fascia and the contiguous lymph nodes, and that in such cases there may be no symptoms whatever of embarrassment of the function of the gland itself. It is then purely a cervical fascia infection.

Whenever an infection is situated in the lower part of the neck, the chest should be carefully examined for connecting reservoirs of pus. Radiographs are in such cases very helpful. When infections in this region follow trauma to the trachea, subcutaneous emphysema is likely to be present. I have not yet observed this complication. Emphysema may be caused by gas producing anaerobes, but until now I have not observed one (Myerson[20]). Infection along the pretracheal fascia into the chest involves the posterior or anterior mediastinum (Case 51). Infection along the prevertebral fascia extends into the posterior mediastinum. Both conditions may be present at the same time.

The blood picture should be carefully watched. This includes daily blood counts and frequent blood cultures. The carotid sheath is close at hand, and a blood stream infection with or without thrombosis is a possibility, though not so common as in other types of neck infection. When a laryngeal edema occurs the question of scarification arises. If the edema is seen early in the course, and if there is not yet severe sepsis and prostration, it probably does no harm if not done deeply. It does not appeal to me strongly, because it does not reach the cause, and possibly does open up new avenues of secondary infection.

There is one type of case which, I believe, makes its appearance frequently in the pediatrics ward, the history of which would read about as follows:

A child, from six months to two years of age, gives a history of an upper respiratory infection, sore throat, coryza, tonsillits, etc., two weeks previously. Frequently, also, swelling of the cervical glands on one or both sides. After a few days the fever subsides, the child feels better, but still not entirely well. Then there is recurrence of the symptoms and the child is admitted to the hospital. Sometimes the patient has been admitted to the hospital before the onset of dyspnea, and sometimes after. The crying voice is somewhat muffled and rough. The cervical glands are again swollen on one or both sides. Such a recurrent history should arouse suspicion that all is not well in the cervical region. There is temperature elevation to between 101 and 105 degrees, and the child refuses food. General examination is negative except for the usual blood pictures of infection.

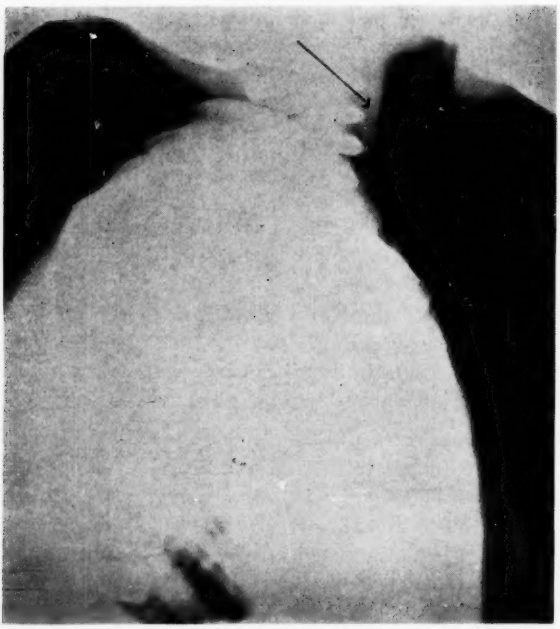


Fig. 7. Lateral neck radiograph showing normal thickness of shadow of retropharyngeal or prevertebral soft parts.

Sometimes examination of the throat of such a child is very difficult and unsatisfactory. Muscular contractions of the pharynx, tongue and palate, presence of frothy secretions, gagging, vomiting and struggling even when mummied and held, make it necessary to be satisfied with a glance or two in place of a prolonged inspection. Under such conditions of muscular contracture, moderate swellings of the pharyngeal walls are indeed difficult to perceive, and the result of the examination is to leave one in considerable doubt. The tonsils may be large with or without crypt exudates. If one side alone is involved, that tonsil may appear to be more prominent than the other. That posterior pillar may be thicker than the other side. The posterior pharyngeal wall may not appear to be swollen. Even digital examination may be negative. Here the lateral neck radiograph is indispensable, for it may in such cases show undis-

putable evidence of thickening of the retropharyngeal tissues. This evidence should never be disregarded. The infection in all such cases which I have seen entered through the tonsil. A pharyngeal infection takes place without definite localization, and eventually involves nearly all of the parapharyngeal tissues, even extending around posteriorly to the opposite side. It is at this time that the characteristic lateral neck radiograph can be obtained. Also, about this time occur the muffled voice shortly followed by dyspnea.

The question which arises is where to drain such a case in which the place of localization is so obscure. When seen before dyspnea is present, I believe that removal of one or both tonsils with or without search for an abscess through the tonsil fossa is rational. If dyspnea is present, I believe that external incision with placing of drains lateral to the larynx on one or both sides, and also placing of a drain into the pharyngomaxillary space close to the tonsil bed on the side showing the cervical adenitis, should be done. If pus is found through the tonsil bed, one may delay external drainage until dyspnea appears. If sepsis and dyspnea appear and do not subside promptly, whether or not pus was found through the tonsil fossa, external drainage should be done, if necessary, on both sides, one drain on the sheath, and one in the pharyngomaxillary space. When the sheath is exposed it is a simple task to explore the side of the larynx. Where there is no actual laryngeal or tracheal obstruction, tracheotomy cannot be expected to relieve the dyspnea. On the contrary, tracheotomy may be harmful. Many have expired with symptoms of asphyxia, with a tracheotomy tube in position. Autopsy findings indicate that lung pathology was already existent, and was the probable cause of these asphyxial symptoms. Infection in the mediastinum may also be expected at the same time. Autopsies on two cases (50 and 51) throw considerable light on these obscure conditions. In one, a child nine months of age, there was marked inflammatory thickening of all the tissues surrounding the hypopharynx, larynx and trachea. The air-way was free. There was a small abscess at the lower pole of one tonsil, found only after a deliberate search for it. In the lungs there was seen, macroscopically, extensive bronchopneumonia with scattered areas of consolidation, and microscopically, extensive bronchiolitis. No report on mediastinum. In the other case, a 56-year-old male, the pathologist reported moderate congestion and edema of the lungs; also acute anterior and superior mediastinitis. In the neck there was a diffuse spreading cellulitis involving the muscles, and the entire prevertebral tissue was edematous and reddish-brown in color. An abscess, extending from the tonsillar fossa down the

lateral wall of the pharynx to the level of the thyroid cartilage, was found. The pathologist noted that the abscess had been opened by intraoral incision. Microscopic examination showed marked pulmonary edema and infiltration of the parenchyma of the kidneys and liver with leucocytes. These autopsy findings are very instructive. Several important deductions can be made. Dyspnea was a prominent symptom in both. In one, the child, there was extensive pneumonia, which was probably the most important cause of death, which was not sudden. In the other, the adult, there was acute anterior and superior mediastinitis and moderate congestion and edema of the lungs, the former probably being the more important cause of death, which was sudden. Both had a small abscess close to the tonsil. These two autopsies support in undeniable terms what Dr. H. P. Mosher has several times stated to the writer in conversation, namely, that in most of such insidious deep neck infections the underlying cause or lesion will be found close to the tonsil. With this I am in absolute agreement, and I am more and more coming to the belief that some of these deep neck infections may be aborted by timely tonsillectomy and a search for such a focus of pus through the empty tonsil fossa. In several instances (Cases No. 29 and 40), I have aborted or cut short a deep neck infection by removal of the tonsils alone. If it fails no particuular harm has been done. In eleven cases the abscess was evacuated through the tonsil fossa (Cases 32, 39, 47, 55, 57, 61, 65, 69, 70, 77, 78). In three of these there was a lead in the form of a granulating tract or fistula through the muscle bed into the pharyngomaxillary space. In six there was no lead, it being necessary to penetrate the bed with a blunt instrument to evacuate the pus. In one of these incision had been made through the lateral pharyngeal wall, posterior to the tonsil, at the time of operation without finding the abscess. On the fifth day after operation the abscess was reached through the tonsil fossa with a blunt instrument. In two a large peritonsillar abscess which extended into the pharyngomaxillary space was evacuated as soon as the upper third of the tonsil was released from its bed.

The dissection in such cases is started with a sharp instrument, liberating the pillar margins only and continued bluntly with a small piece of rolled gauze in the grasp of a clamp, after which the snare is applied. The postoperative healing is practically the same as after ordinary uncomplicated tonsillectomy. Cervical gland swelling immediately subsides, and the patient is relieved of the discomfort existing prior to the operation. A peritonsillar abscess deeply situated behind and perhaps below the tonsil fossa cannot

be drained by simple incision through the upper part of the anterior pillar. In any case, deep incision through the lateral and lower part of the anterior pillar is very risky, and removal of the tonsil is safer and more effective. In advocating this I know that I am on controversial ground. Not to be misunderstood, I wish to state that I am not advocating that this procedure should supplant simple incision when such will effectively drain the abscess.

Occasionally trauma from an unmasticated bolus of food, or from a fish bone or chicken bone will cause an infection lateral to the hypopharynx. If there is prompt localization it may sometimes be drained by incision through the laryngeal or esophageal speculum. The head should be low in position, and suction simultaneously applied to avoid inspiration of pus into the trachea and lungs. Incision through endoscopic instruments is rational if one is sufficiently skillful in their use. I do not believe that trauma to the hypopharynx is nearly so dangerous as trauma to the esophagus, farther down. Myerson[20] has reported infectious emphysema in these traumatic cases.

Summarized, the possible alarming and fatal complications of infection along the pretracheal fascia are: edema of the larynx, pneumonia, empyema, mediastinitis and blood stream infection. The last mentioned is not so common as with infections in the pharyngomaxillary and submaxillary regions.

PREVERTEBRAL FASCIA INFECTION

The typical example of infection in this location is the midline retropharyngeal abscess which occurs most frequently in infants and young children. The symptoms and signs of frank retropharyngeal abscess located in the midline are well known. It is caused by infections high up in the pharynx, in the adenoid, nasal mucosa, and uppermost retropharyngeal lymph nodes. Infection following necrosis of the cervical vertebrae is not at first an infection of this space, but may involve it secondarily. In such cases it may travel laterally, pointing into the posterior cervical triangle. High up it would at first be found behind the great vessels in the retrostyloid part of the pharyngomaxillary space.

One of the first symptoms is refusal of the child or infant to take food. At the same time there is moderate elevation of temperature, and the patient gives evidence of discomfort. There may be slight neck rigidity. As the infection develops and swelling of the postpharyngeal tissues appears, there is a muffling of the voice,

Fig. 8. Lateral neck radiograph showing abnormal thickness of shadow in presence of retropharyngeal abscess. Barium mixture in pharynx.

gutteral in character, and later a type of noisy breathing similar to that heard when a hot morsel of food is held in the back of the mouth while awaiting sufficient cooling for swallowing. If the condition is not seen and recognized at this time, and spontaneous rupture does not occur, embarrassment of respiration increases. The clinical picture then has become grave for several reasons. Usually by this time, because of refusal to swallow, the patient has developed a marked degree of dehydration, which, with sepsis and embarrassment of respiration, has brought about considerable exhaustion. The first requisite then is to relieve the respiratory obstruction. This obstruction is caused by capping of the larynx by the swelling of the posterior pharyngeal wall. This swelling quickly disappears if the abscess is incised and drained. If the dyspnea is so severe that any exertion tends to cause asphyxia, a preliminary tracheotomy

must first be done. If one is sufficiently skillful with endoscopic instruments the abscess may be evacuated through these. There is, no doubt, possibility of rupturing the abscess when doing this. The ideal time to drain a retropharyngeal abscess is before it has reached the level of the larynx, while it can be seen and reached through the mouth. The head should be low, and suction at hand. These midline retropharyngeal abscesses tend to gravitate into the posterior mediastinum along the fascia of the longus colli muscles. I have never observed spontaneous rupture, though, no doubt, it occasionally occurs. I have also not seen one which started in midline, take a lateral course, though it is conceivable that it might travel along the fascia of the scalenus anticus. In this case it would reach the carotid sheath which crosses this muscle. It might then extend into the posterior cervical triangle in the lower part of the neck or into the axilla and costo-caracoid regions.

If peroral incision is made before respiratory embarrassment has occurred, recovery is usually prompt and permanent. External drainage may be accomplished by Dean's[24] approach. This is strongly indicated in those cases where there is dyspnea, for the evacuation of the abscess would immediately relieve it, and would probably avoid a tracheotomy. It is sometimes impossible to make a satisfactory examination in such cases, because the struggling while being examined increases the dyspnea. It is thus very injudicious to use any force. Happily, a lateral neck radiograph gives invaluable aid, as it will always show definite evidence, particularly if the abscess has not already evacuated itself. Where there is no dyspnea, digital examination of the pharyngeal wall may be employed to supplement visual examination. Digital examination has not been as helpful to me as I have desired, especially in cases where the visible evidence has been doubtful. Here again the lateral neck radiograph is very helpful. A widening of the shadow of the tissues between the vertebral bodies and the pharyngeal cavity should never be ignored. A review of the history and findings, including radiograph, of cases Nos. 44 and 63, and a study of the autopsy specimen of case No. 50, emphasizes this. The radiograph should be given at least as much consideration as inspection and palpation, if not more.

If an infection high up near the skull base is in a lateral position at the start it becomes a parapharyngeal, or pharyngomaxillary, infection, and in this paper is placed in that group. It is possible that both the midline and the lateral regions may be involved at the same time. In such a case peroral incision alone would probably not be conclusive, and if the sepsis persisted, external drainage of

the pharyngomaxillary space would be indicated. The evidence in one of the aforementioned cases (No. 50) indicates that sometimes in these obscure cases where there is little or no external neck swelling the infection eventually involves all the fascial regions. In this the neck radiograph gave antemortem evidence of infection of the prevertebral fascia, and the autopsy gave evidence of infection of the pretracheal and buccopharyngeal fasciæ. It also indicates that primary infections of other compartments, such as the pharyngomaxillary, starting from a small abscess in relation with the tonsil, and secondary to a tonsillitis, can and do involve the retropharyngeal space not with abscess formation, but with a diffuse inflammatory thickening which gives evidence of itself in the lateral neck radiograph. Inspection and palpation through the mouth are unsatisfactory in such cases. They are apt to have dyspnea without laryngeal edema. It seems that, in cases of retropharyngeal abscess, while the abscess gravitates and heads for the posterior mediastinum from the start, death is more likely to be caused by the mechanical laryngeal obstruction or by aspiration pneumonia after rupture of the abscess, or by both.

CERVICAL GLAND INFECTION

There are two principal types of cervical gland infection. The one involves chiefly the glands of the superficial chain, and the other involves the glands of the deep chain. Clinically this distinction cannot be too strongly stressed. The superficial glands lie between the superficial fascia and the superficial layer of the deep fascia, and are therefore in close proximity to the overlying skin. They are particularly numerous along the external jugular vein and its tributaries. They are found under the skin of the entire cervical region, and drain into the deep chains. The deep chains are arranged in certain, more or less definite, group chains, some chains horizontal, others vertical or oblique, and others irregularly. They are situated at all fascial depths, i. e., all layers of the deep fascia are plentifully studded with lymphatics.

High up in the retropharyngeal space there are several lymph nodes which take drainage from the region of the nose and nasopharynx. When these suppurate a retropharyngeal abscess is the result. In a more or less horizontal line from the occiput to the chin there are several groups of nodes, namely, the suboccipital, the postauricular or mastoid, the preauricular and parotid, the submaxillary and the submental. In the posterior cervical triangle there are glands in close series with the sheath glands. They extend from below the occipital and mastoid bones to the clavicle and scapula.

Those in the parotid group are found on the fascia of the gland, and are also enmeshed in the parotid gland structure. There is very little doubt that swelling in this region, closely resembling mumps, may be caused entirely by inflammatory swelling of these lymph glands alone. It is said that all the nodes in relation with the submaxillary gland are in relationship with its capsule alone, and none are enmeshed in its structure (Trotter[5]). The submental glands lie in the submental triangle below the chin, and receive drainage from the lower lip and chin. The postauricular group nodes lie superficial to the mastoid bone, and are frequently seen when performing mastoid operations. There are some rather important lymph nodes in and about the thyroid gland fascia and along the recurrent laryngeal nerve. There is also a superficial chain along the external jugular vein; also one along the anterior jugular vein. The glands in the submaxillary region receive the lymph drainage from the tongue, floor of the mouth and lower teeth. These lymph channels turn sharply in the lateral direction above the level of the hyoid bone to reach glands on the corresponding side of the neck which are part of the chain along the carotid sheath. The nodes on the submaxillary salivary gland receive some of this drainage from the teeth (Blassingame[13]). I have seen an abscess form in glands at the anterior border of the sternomastoid muscle just below the angle of the jaw, from infection in the lower dental region by way of the buccal side, and without involvement of the intervening tissues. In other words, what may be designated as a lymphatic jump.

Receiving the drainage from all lymphatics of the head and neck is the large, rich, deep chain situated along the carotid sheath beneath the sternomastoid muscle, and arranged in a more or less vertical direction. This is the most important lymphatic group in the neck. When inflamed they become quite adherent to the fascial sheath of the vessels, and it is not at all unreasonable to assume that blood stream infection frequently occurs in this way.

A knowledge of the applied anatomy of the cervical lymphatics is very helpful to an understanding of the behavior of cervical adenitis. There is a considerable clinical distinction between inflammation involving the superficial, and inflammation involving the deep glands.

INFLAMMATION OF THE SUPERFICIAL GLANDS

The sepsis is not so severe, and is of short duration. There is a greater tendency to abscess formation, and when this occurs it is superficially located, subcutaneous. Fluctuation occurs frequently, and is readily elicited. Inflammation of the skin, manifested by

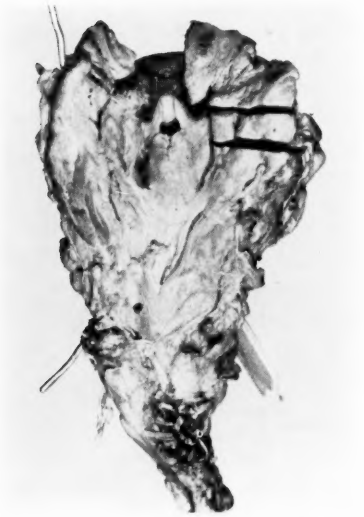


Fig. 9. Specimen removed at autopsy from Case No. 50. There is evident inflammatory thickening of all the tissues surrounding the hypopharynx, larynx and trachea. The air-way was free, though the child had severe dyspnea. There was a small abscess at the lower pole of one tonsil which was not known to be present before death. There was bronchopneumonia with scattered areas of consolidation.

redness, often occurs. In the absence of severe sepsis, and in the absence of involvement of the deep lymphatics, it is justifiable and wise to wait for fluctuation; in fact, it is the only type of neck infection where that can be stated. There is no involvement of the fascial plane except at the point of suppuration. There may be spontaneous subsidence without suppuration. There may be extension to the deep chain with resultant increase in the severity of the infection. When this occurs the condition should be regarded with all the caution that applies to a fascial plane infection. Even after a superficial abscess has been incised and drained this extension may occur.

There is no justification for the use of the aspirating needle, as fluctuation is recognizable as soon as abscess formation occurs, in which event the scalpel is indicated. Aspiration with a needle is apt to produce a widely spreading cellulitis where none existed

previously, and when there is suspected abscess of the deep chain, exploration with the needle is even more objectionable for the same reason, and also because of the greater danger of perforating deeply lying blood vessels. In these, edematous pitting can often be elicited.

INFLAMMATION OF THE DEEP GLANDS

In these the sepsis is more severe. Suppuration occurs later, and does not so often take place. Fluctuation is therefore much less commonly to be elicited, but pitting on deep pressure with the finger is often present when there is no fluctuation. Often the individual glands cannot be felt, and there is only a slight fullness along the sternomastoid muscle or a diffuse prominence on that side of the neck. There is frequently spontaneous subsidence of such symptoms and signs, with recovery. This is frequently observed by the pediatrist. The condition may remain more or less stationary for several weeks, and suddenly change into an alarming status by extension and the development of a most profound sepsis. Blood stream infection with distant complications may occur suddenly and without warning. Constant, close watching is necessary. A severe chill is sometimes the first sign.

When infection strikes into the neck from the portal of entrance in the upper respiratory areas, the lymphatic system bears the brunt of the initial invasion, and gives the first visible evidence. In this early period there is usually a sustained elevation of temperature. Later, if the temperature curve is characterized by abrupt rises and drops, especially if accompanied by chills, the blood stream has been invaded. Sometimes this invasion of the lymphatics of the neck occurs with direct invasion of the tissues of the neck by continuity from the portal of entrance, as, for example, the teeth. In other instances there is a lymphatic jump from the portal of entrance through lymph vessels directly to the lymph nodes of the neck, across fairly large areas of uninvolved tissues. I have observed this to occur from the pharyngeal mucosa and from the buccal side of the alveolar process of the mandible. It occurs most frequently from the tonsil.

Usually when there is improvement in the gland inflammation it is of favorable prognostic significance, other features not indicating the reverse. Observance of the behavior of the lymphatic inflammation is of the utmost importance. It happens sometimes that the inflammation at the portal of entrance subsides completely, leaving the lymph node inflammation as the only remaining evidence of the infection. This happens frequently after tonsillitis. After

a fairly long delay of this nature a deep fascia involvement may still occur. Sometimes deep infection along the fascial planes is not accompanied by visible involvement of the lymphatics. It is in such cases that there is little or no visible swelling of the neck. Infection of the pretracheal, or visceral, fascia is often present without swelling which can be recognized.

The nodes in the neck that are most frequently involved are those situated at the anterior border of the sternomastoid muscle just below the angle of the jaw. They lie about over the bifurcation of the carotid artery. I have observed that infections entering through the pharynx and tonsils very frequently cause inflammatory swelling of the glands below the jaw angle, and that almost immediately. This is a typical lymphatic jump. There is usually high temperature. Infections in the submaxillary region differ in that this region swells, becomes very much indurated, and the individual glands cannot usually be palpated. With infection in the pretracheal fascia lymph node swelling is not so common, and often none is palpable. Occasionally there is a lymphatic jump from infection in and about the teeth, especially when occurring in children. In such cases the glands may break down and form an abscess. When there has been inflammatory swelling of glands for more than several weeks an abscess frequently results, but there may, on the other hand, occur spontaneous subsidence of the inflammation even in such prolonged cases. Glands which have been swollen for several weeks or more may undergo late suppuration with the formation of an abscess, without the development of high fever. If such an occurrence is capable of being determined, by fluctuation or pitting, it should be drained.

The important thing to determine when there are inflamed glands is—where the infection entered. If seen early and during the initial attack this evidence will be found somewhere in the upper respiratory tract, teeth or ear mechanism. If seen later the original inflammation in these parts may have disappeared. A red throat should never be forgotten or ignored in evaluating the history.

The following is a resume of my conception of the management of acute cervical adenitis:

1. With involvement of the superficial glands chiefly, it is my custom to wait for localization and abscess formation, providing there is no sepsis. As soon as an abscess forms, as manifested by the usual signs, incise and drain.

2. If no abscess forms and there is no sepsis, wait. In such cases there may occur spontaneous subsidence.

3. If the deep glands become involved, wait for signs of abscess formation, but be on the alert for sepsis and fascial plane involvement. If sepsis and extension occur, drain. At first, when the inflammation is chiefly lymphatic, there is usually a sustained high temperature. Later, when sepsis intervenes, there will be abrupt rises and drops in temperature and perhaps chills.

When this occurs, drainage is indicated. Positive blood culture indicates ligation or resection of the internal jugular vein. With chills and sweats and negative blood culture it is safer to drain.

4. The deep glands may remain enlarged for weeks, and if there is no sepsis, drainage may be postponed. Many of these recover spontaneously.

5. In cases where severe sepsis and distant complications indicate that a blood stream infection is present, and that drainage and jugular ligation must be done, the inflamed, swollen glands on the sheath should be carefully excised, if only to afford proper access to the sheath. In such instances dividing the belly of the omohyoid greatly facilitates the operative procedure.

It should be remembered that any infection of the head or neck which is the source of infection in the glands can thereby be the cause of a deep neck infection. In the vast majority of cases the trouble will be found in or near the tonsil, and the remedy is obvious. When to remove the tonsils is often a controversial question. In thirteen cases (29, 32, 39, 40, 47, 55, 57, 61, 65, 69, 70, 77 and 78) I have removed the tonsil at the time. In properly selected cases it is thus possible to drain a neck infection. In such cases there is usually an immediate subsidence of the gland infection, usually in two to four days. Where there is frank primary acute tonsillitis, tonsillectomy is never to be considered. Where, however, there is peritonsillar inflammation, especially where there is abscess formation, it may be considered. Iglauer[21] has recommended that where there is neck infection requiring drainage, this be done, and that the tonsils be removed at the same time. A peritonsillar abscess in child or adult, which has existed longer than the usual intense acute stage of involvement, sometimes becomes encapsulated and lined with granulation tissue. In these there is frequently a fistulous communication with the same character of lesion in the neighboring parapharyngeal area, and such a condition is cured by tonsillectomy. Any cervical gland inflammation which may have

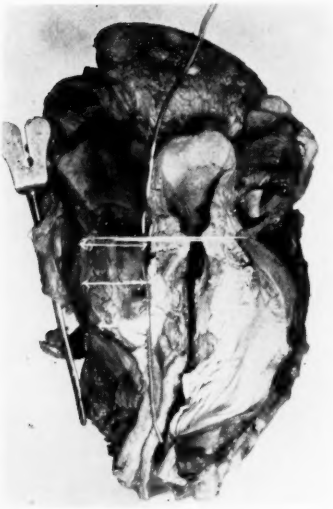


Fig. 10. Specimen removed at autopsy from Case No. 51. This disclosed a diffuse spreading cellulitis involving the muscles, and the entire prevertebral tissue was edematous and reddish-brown in color. An abscess, extending from the tonsillar fossa downward along the lateral wall of the pharynx to the level of the thyroid cartilage was present. Peroral incision of this peritonsillar abscess had been done and was noted by the pathologist. This drainage was inadequate.

been present immediately subsides unless there is already abscess formation in the glands.

ANESTHESIA

The question of anesthesia for operation requires serious consideration. First of all, the anesthetist must be an expert. It must surely be obvious that where there is actual dyspnea, general anesthesia is out of the question until the airway has been completely restored. Where the encroachment is limited to the tongue and pharyngeal region, and where this can be completely corrected by the insertion of a pharyngeal breathing tube, the use of a general anesthesia is safe. Where the dyspnea is caused by edema of the larynx or by pathology in the deep respiratory tract, a general anesthetic of any kind should never be administered. Unconsciousness

should, under such circumstances, never be induced, and narcotics should not be administered. After tracheotomy, general anesthesia, including ether or chloroform, may be administered if there is no involvement of the lungs. If the dyspnea is well established and severe, there may not be time even to infiltrate a local anesthesia. For intratracheal administration, introduction of a tube or catheter through a tracheal opening is preferable to introduction through the mouth and an edematous inflamed larynx. In that large percentage of cases where there was no dyspnea at any time, general anesthesia was most successfully administered in all of my cases. In all those where there are no contraindications we use routinely intrapharyngeal with nitrous oxide and oxygen mixture, cyclopropane, avertin and even ether, in various combinations. Where pulmonary involvement is present or suspected, ether or chloroform should not be administered. In such cases we use the nitrous oxide and oxygen mixture, and at the conclusion of the operation, stimulate respiration with carbon dioxide and oxygen, combined or separately. In cases with marked submaxillary and sublingual swelling, with crowding and swelling of the tongue, we have also had complete success with general anesthesia, providing a breathing tube could be inserted into the pharynx. Where this is not possible I should consider inserting a catheter of the intratracheal type, but smaller, through each nostril. If this is not possible, tracheotomy is justifiable. In none of our cases have we been unable to insert a breathing tube through the mouth. Unless the mouth tube is used there is great danger of what Chevalier Jackson described as "lingual asphyxia." The Mosher life-saver laryngeal canula or tube can be very valuable in such instances. With evipal I have had no experience.

When there is dyspnea, sedatives and narcotics should not be administered for any reason until the dyspnea has been completely relieved.

Now as to local anesthesia, I can imagine late cases with severe dyspnea and exhaustion, and in a moribund condition, where no anesthesia at all, local or general, should be administered. In those where there is no dyspnea, and where conditions are not present which would contraindicate general anesthesia under any circumstances, local anesthesia need not be considered except by voluntary preference. If there is no inflammatory involvement of the parts, a good local anesthesia may be easily accomplished. The cervical plexus must be reached at the posterior border of the sternomastoid muscle. If, however, there is inflammatory swelling of the part to be in-

jected, it is extremely difficult to obtain anesthesia of anything but the skin. It is then only partial anesthesia. I myself once had a cervical gland abscess drained after local skin anesthesia, and can testify that the skin anesthesia was perfect, but that the pain on reaching the gland was as severe as when a heavy automobile door was shut on my finger. The pain incident to the extraction of a molar teeth without anesthesia is as nothing by comparison. This I have also experienced. If one is skillful enough to make successful paravertebral injections of the cervical nerves at their points of exit from the cervical column, the ideal anesthesia is obtainable. In most instances it will be necessary to use the posterior approach through thick muscle layers, and a high degree of skill is required. By this route one avoids traversing infected areas. It is conceivable that deep insertions of the injecting needle into the swollen, infiltrated areas, even though subcutaneous throughout, may carry infection into tissues until then uninvolved, though this may not be appreciated because of the immediate drainage incisions and the resultant postoperative tissue reaction. A tracheotomy should always be done under local anesthesia or without any anesthesia. I use local anesthesia in deep neck infections only when conditions are present which make general anesthesia hazardous.

SUMMARY AND CONCLUSIONS

1. Seventy-eight cases of deep neck infection have been studied and analyzed.

2. The symptomatology, diagnosis and management have been given emphasis.

3. A classification based on anatomical regional involvement has been suggested.

4. Analysis of the material corroborates previous conclusions that the most frequent portal of entrance of deep neck infection is in and about the tonsils, and that the most frequently present invader is the streptococcus hemolyticus.

5. Phlebitis of the internal jugular vein, with or without thrombosis, should be anticipated and promptly recognized in all cases of deep neck infection.

6. The problem of anesthesia is discussed.

421 Huguenot St.

REGIONAL PATHOLOGY

Cases	Age and Sex	Etiology Portal of Entrance	Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 1 Jennie R.	9 mo. F.	Nasopharyngitis Infected adenoid	....	.....	.....	Retro-pharyngeal abscess	.....	.....	None	Per-oral incision	Recovery
No. 2 Rose A.	8 mo. F.	Infected tonsils and adenoid	....	.....	.....	Retro-pharyngeal abscess	.....	.....	None	Per-oral incision	Recovery
No. 3 Joseph H.	6 yrs. M.	Infected tonsils and adenoid	....	.....	.....	Retro-pharyngeal abscess	.....	.....	None	Per-oral incision	Recovery
No. 4 Mary F.	42 yrs. F.	Hypo-pharynx trauma	....	.....	.....	.....	Bucco-pharyngeal abscess	.....	None	Incision through laryngo-scope	Recovery
No. 5 Oscar B.	19 mo. M.	Infected tonsil	Lateral pharyngeal abscess	.....	.....	.....	.....	.....	None	Per-oral incision	Recovery
No. 6 Kath. R.	20 yrs. F.	Pharyngitis and infected tonsils	Pharyngo-maxillary cellulitis	.....	.....	.....	.....	.....	None	Non-surgical treatment	Recovery
No. 7 Center H.	14 mo. M.	Infected tonsils and adenoid	Pharyngo-maxillary abscess	Submax-illary and parotid	Secondary	.....	.....	.....	Rupture into external auditory canal	External drainage	Recovery
No. 8 Leonard P.	16 yrs. M.	Infected tonsil remnant	Pharyngo-maxillary abscess	.....	Secondary	.....	.....	Sheath infection	Thrombosis of internal jugular vein and cavernous sinus	Per-oral incision	Death
No. 9 Gilbert K.	3 yrs. M.	Infected tonsils	Pharyngo-maxillary abscess	.....	Secondary with abscess	.....	.....	.....	Lymph glands abscess	External drainage	Recovery
No. 10 Jane M.	5 yrs. F.	Pharyngitis and infected tonsils	Pharyngo-maxillary abscess, secondary	Submax-illary and parotid Infection, secondary	Cervical glands abscess, primary	.....	.....	.....	None	External drainage	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	REGIONAL PATHOLOGY Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 11 Shaw C.	17 yrs. M.	Pharyn-gitis	Pharyngo-maxillary abscess	.....	.....	.....	.....	.....	Secondary hemorrhage Paresis of the 9th, 10th and 11th cranial nerves	External drainage	Recovery
No. 12 Sally H.	4 yrs. F.	Trauma from adenoid operation	Pharyngo-maxillary abscess	.....	Glands on sheath infected	.....	.....	Infected	Phlebitis of internal jugular	External drainage and resection of jugular vein	Recovery
No. 13 Dorothy S.	19 yrs. F.	Otitis media and petrous temporal bone infection	Pharyngo-maxillary abscess	Parotid fascia infection	.....	.....	.....	.....	None	External drainage	Recovery
No. 14 Rose F.	32 yrs. F.	Infection in zygo-matic fossa following local an-aesthesia injection	Pharyngo-maxillary abscess	Parotid and submaxil-lary fascia infection	.....	.....	.....	.....	None	External drainage	Recovery
No. 15 Edythe R.	26 yrs. F.	Tonsil remnant	Pharyngo-maxillary abscess	Submaxil-lary space abscess Primary	.....	.....	.....	Carotid sheath infection	None	External drainage	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	REGIONAL PATHOLOGY Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 16 Alfred S.	7 yrs. M.	Rhino-pharyn-gitis	Pharyngo-maxillary abscess	Submaxil-lary space infection	Lymph-adenitis, secondary	.....	.....	.....	Mastoiditis Sigmoid sinus thrombosis Hip joint abscess Paresis of 12th nerve Paresis of pupillary fibers of cervical sympathetic nerve	Ext. neck drainage Mastoid-ectomy Drainage of sigmoid sinus Resection of int. jug-ular vein Hip joint drainage	Recovery
No. 17 Eliz. W.	6 yrs. F.	Pharyn-gitis	.....	.....	Cervical gland abscess	.....	.....	.....	None	External drainage	Recovery
No. 18 Joseph C.	10 mo. M.	Infected tonsils and adenoid	.....	.....	Cervical gland abscess	.....	.....	.....	None	External drainage	Recovery
No. 19 Sylvia F.	14 yrs. F.	Trauma from tonsil operation	Small local-ized ab-scess be-neath ton-sil fossa muscle bed	.....	.....	.....	.....	.....	None	Spontaneous drainage	Recovery
No. 20 Edgar W.	21 yrs. M.	Dental infection	.....	Submaxil-lary fossa infection Parotid fascia infection	.....	.....	.....	.....	None	External drainage	Recovery
No. 21 Charity D.	53 yrs. F.	Thyroid gland fascia lymph node	.....	.....	Lymph-adenitis on visceral fascia	.....	Pretracheal or visceral fascia abscess	.....	None	External drainage	Recovery

REGIONAL PATHOLOGY

Cases	Age and Sex	Etiology Portal of Entrance	Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 22 Daniel M.	30 yrs. M.	Dental infection	.....	Submaxillary and submental triangle infection, with infection of the floor of the mouth	.....	.....	.....	.....	None	External drainage	Recovery
No. 23 Rose L.	7 yrs. F.	Dental Alveolar abscess	.....	Submaxillary and submental triangles Infection floor of mouth	.....	.....	.....	.....	None	External drainage	Recovery
No. 24 Louise B.	47 yrs. F.	Pharyngitis	.....	Submaxillary and parotid fascia, and carotid sheath	Secondary Without suppuration	.....	.....	Carotid sheath infection	Thrombosis of internal jugular vein Thrombosis of veins of both legs with abscess formation Pelvic abscess Bilateral choked disc Cystitis	External drainage of carotid sheath Leg abscess drainage bilateral Pelvic abscess drainage	Recovery
No. 25 Raymond C.	32 yrs. M.	Tonsil remnant	Pharyngo-maxillary infection	.....	Secondary	.....	.....	.....	None	Per-oral incision	Recovery
No. 26 Ada F.	31 yrs. F.	Pharynx and Fauces—Tonsils out	Pharyngo-maxillary infection	.....	Hemorrhagic inflamed glands on sheath	.....	.....	Carotid sheath infection	None	External drainage	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	REGIONAL PATHOLOGY Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 27 Francis F.	4½ ys. M.	Through nasal and pharyngeal mucosa Tonsils out	Pharyngo-maxillary infection	.....	Inflamed glands on sheath	.....	.....	Carotid sheath infection	Inflammation in calf of right leg Positive blood culture	External drainage	Recovery
No. 28 Louis P.	3 yrs. M.	Through tonsils and pharynx	Pharyngo-maxillary infection	.....	Cervical gland abscess, late	.....	.....	.....	None	Tonsillectomy and external drainage of late cervical gland abscess	Recovery
No. 29 Clive P.	4 yrs. M.	Through tonsils and pharynx	.....	.....	Deep cervical chain lymph-adenitis, bilateral	.....	.....	.....	Chills, etc.	Tonsillectomy	Recovery 48 hours
No. 30 Teresa G.	30 yrs. F.	Dental, following extraction	.....	Submental, submaxillary and parotid regions, and floor of mouth	.....	.....	.....	.....	Osteomyelitis of mandible	External drainage	Recovery
No. 31 Frances S.	21 yrs. F.	Through pharynx Tonsils out	Pharyngo-maxillary above	.....	.....	.....	Buccopharyngeal Lower	.....	Facial Erysipelas five days after discharge	External drainage	Recovery
No. 32 Roy W.	3 yrs. M.	Tonsil	Pharyngo-maxillary Retrostyloid	.....	Lymph-adenitis, secondary	.....	.....	.....	None	Tonsillectomy	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	REGIONAL PATHOLOGY Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Viscera! Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 33 Betty Ann B.	10 yrs. F.	Tonsil	Pharyngo-maxillary and parotid compart-ments	.....	Mild secondary	.....	.....	.....	None—spontaneous rupture into pharynx and into ear canal	Non-surgical	Recovery
No. 34 James T.	7 yrs. M.	Tonsil	Pharyngo-maxillary and parotid	Submaxil-lary com-partments	Mild secondary	.....	.....	.....	None	External drainage	Recovery
No. 35 Guette H.	29 yrs. F.	Dental infection No extrac-tion	.....	Submaxil-lary, sub-mental and parotid regions	.....	.....	.....	.....	None	External drainage	Recovery
No. 36 Mary P.	4 yrs F.	Pharynx and fauces Tonsils out	Pharyngo-maxillary infection	Submaxil-lary adeno-pathy	Secondary	.....	.....	Carotid sheath infection	Thrombosis of internal jugular vein	External drainage and ligation	Recovery
No. 37 Lawrence M.	5 yrs. M.	Through tonsil Vincent's ulcer	Pharyngo-maxillary infection	.....	Secondary with post-operative sloughing and hem-orrhage	.....	.....	Carotid sheath infection	Positive blood culture Secondary venous hemorrhage Pharyngeal fistula Pneumonia Empyema	External neck drainage Thora-cotomy	Recovery
No. 38 Warren G.	13 yrs. M.	Pharynx and tonsils	.....	.....	Superficial abscess	.....	.....	.....	None	External drainage	Recovery
No. 39 Lee R.	35 yrs. M.	Tonsil Peritonsil-lar abscess	Para-pharyngeal abscess	.....	Lymph-adenitis, secondary	.....	.....	.....	None	Tonsillec-tomy and drainage through tonsil fossa	Recovery

REGIONAL PATHOLOGY

Cases	Age and Sex	Etiology Portal of Entrance	Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 40 Jack B.	38 yrs. M.	Tonsil and pharynx	Retrostyloid compartment Posterior cervical triangle	.....	.....	.....	Mild Right side of hypopharynx and epiglottis edematous	.....	None	Tonsillectomy	Recovery
No. 41 Claudia S.	21 yrs. F.	Pharynx and nose Tonsils out	Parapharyngeal infection	.....	.....	.....	.....	.....	None	Non-surgical	Recovery
No. 42 John D.	81 yrs. M.	Mouth and pharynx—Tonsils not out	.....	.....	Cervical gland abscess	.....	.....	.....	None	External drainage	Recovery Death from senility later
No. 43 Anna Di B.	46 yrs. F.	Dental after extraction	Pharyngo-maxillary	.....	.....	.....	.....	.....	None	Non-surgical	Recovery
No. 44 Josephine Di B.	17 mo. F.	Pharynx and tonsils	Pharyngo-maxillary, right	.....	Cervical gland abscess, bilateral	.....	Pretracheal fascia, manifested by hoarseness, dyspnea and slight cyanosis	.....	Broncho-pneumonia, right upper	External drainage	Recovery
No. 45 Helen N.	20 yrs. F.	Base of tongue and lingual tonsil	.....	Submental triangle Ludwig's angina	.....	.....	.....	.....	None	External drainage	Recovery
No. 46 Eleanor E.	19 yrs. F.	Tonsil remnants and pharynx	.....	.....	Secondary cervical gland abscess	.....	Pretracheal fascia infection	.....	Edema of one side of larynx and hypopharynx. Sequel—meningitis	External drainage	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
					REGIONAL PATHOLOGY						
No. 47 Josephine R.	10 mo. F.	Peritonsillar abscess	Pharyngo-maxillary infection	.....	Lymph-adenitis	.....	.....	.....	None	Homo-lateral tonsillectomy	Recovery
No. 48 David G.	8 yrs. M.	Peritonsillar abscess	Pharyngo-maxillary infection	.....	Secondary	.....	.....	.....	None	Spontaneous recovery Non-surgical	Recovery
No. 49 John C.	21 yrs. M.	Dental, after extraction	.....	Submaxillary and submental triangles	.....	.....	.....	.....	Sequelae—mumps, orchitis and pancreatitis	External drainage	Recovery
No. 50 Chas. F.	9 mo. M.	Peritonsillar abscess found at autopsy	Para-pharyngeal abscess found at autopsy	.....	.....	.....	Pretracheal fascia infection	.....	Broncho-pnemuonia with scattered areas of consolidation	Per-oral incision Inadequate External drainage	Death
No. 51 Rosario G.	56 yrs. M.	Peritonsillar abscess	Para-pharyngeal abscess found at autopsy	.....	.....	.....	Pretracheal fascia abscess found at autopsy	.....	Anterior and superior mediastinitis Edema of lungs	Per-oral incision	Death
No. 52 Margaret J.	20 yrs. F.	Dental after extraction	Pharyngo-maxillary abscess from mandibular nerve injection	Submaxillary infection from the alveolar process	.....	.....	.....	.....	None	External drainage	Recovery
No. 53 Carolyn Mc.	4 yrs. F.	Pharynx, tonsils and nose	.....	.....	Superficial cervical gland abscess	.....	.....	.....	None	External drainage	Recovery

REGIONAL PATHOLOGY

Cases	Age and Sex	Etiology Portal of Entrance	Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 54 John M.	3 yrs. M.	Pharynx and tonsils	.....	.....	Superficial cervical gland abscess	.....	.....	.....	None	External drainage	Recovery
No. 55 Jane E.	3½ yrs. F.	Pharynx and tonsils	Pharyngo-maxillary abscess	.....	Secondary lymph-adenitis	.....	.....	.....	None	External drainage	Recovery
No. 56 Fred G.	6½ yrs. M.	Pharynx and tonsils. Scarlet fever	.....	.....	Cervical gland abcess with discharging sinus	.....	.....	.....	Scarlet fever developing after drainage operation	External drainage	Recovery
No. 57 Paul P.	4 yrs. M.	Tonsil	Pharyngo-maxillary abscess	.....	Secondary lymph-adenitis	.....	.....	.....	None	Tonsillectomy and per-oral drainage through tonsil fossa	Recovery
No. 58 Grace S.	5 yrs. F.	Dental, after filling of cavity	.....	Submaxillary infection	Cervical gland abscess, deep chain	.....	.....	.....	None	External drainage	Recovery
No. 59 Patricia S.	4 yrs. F.	Pharynx and tonsils	.....	.....	Cervical adenitis with late abscess formation	.....	.....	Secondary to cervical adenitis	None	External drainage	Recovery
No. 60 Francis C.	5 yrs. M.	Dental	.....	Submaxillary space infection	Cervical gland abscess	.....	.....	.....	None	External drainage	Recovery
No. 61 Barbara B.	9 yrs. F.	Tonsils	Pharyngo-maxillary abscess	.....	Secondary cervical adenitis	.....	.....	.....	None	Tonsillectomy Per-oral incision through tonsil fossa	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	REGIONAL PATHOLOGY Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 62 George E., Jr.	2 yrs. M.	Dental	.....	Submax-illary abscess	.....	.....	.....	.....	None	External drainage	Recovery
No. 63 William D.	5 yrs. M.	Tonsils and pharynx	Pharyngo-maxillary abscess, retro-styloid compart-ment	.....	Secondary No abscess	.....	.....	Thickened inflamed sheath	None	Internal and external drainage	Recovery
No. 64 Agnes A.	9 yrs. F.	Dental, buccal side	.....	Submax-illary abscess	.....	.....	.....	.....	None	External drainage after spontan-eous ruptue	Recovery
No. 65 Nancy P.	4 yrs. F.	Tonsils and pharynx	Pharyngo-maxillary abscess	.....	Secondary No abscess	.....	.....	.....	None	Internal drainage through tonsil fossa after ton-sillectomy	Recovery
No. 66 Norman G.	5 yrs. M.	Mouth trauma in gingival area	.....	Submax-illary abscess	.....	.....	.....	.....	None	External drainage	Recovery
No. 67 William R.	32 yrs. M.	Dental, following extraction	.....	Submaxil-lary and submental with infra-mylohoid abscess	.....	.....	.....	.....	Osteomyelitis of mandible	External drainage and multiple dental ex-tractions	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	REGIONAL PATHOLOGY Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 68 Carolina Di I.	34 yrs. F.	Dental infection, maxillary, following extraction	Zygomatic and temporal fossae abscess Pharyngo-maxillary infection without abscess	.....	.....	.....	.....	.....	Mild broncho-pneumonia, right base. Necrosis of maxilla with sequestration	External drainage Ligation ext. carotid Caldwell-Luc. left	Recovery
No. 69 Nancy P.	4 yrs. F.	Tonsil—Peritonsillar abscess	Pharyngo-maxillary abscess	.....	Secondary No abscess	.....	.....	.....	None	Tonsillectomy with exposure of abscess	Recovery
No. 70 John F., Jr.	11 yrs. M.	Tonsil remnant	Pharyngo-maxillary abscess	.....	Secondary No abscess	.....	.....	.....	None	Through tonsil fossa, after removal of remnant	Recovery
No. 71 Marilyn F.	8 yrs. F.	Pharynx and nose	Pharyngo-maxillary inflammation No abscess	.....	Cervical gland abscess Superficial and deep	Retro-pharyngeal inflammation No abscess	.....	.....	None	External drainage	Recovery
No. 72 Roy LeB.	5 yrs. M.	Infected tonsils	Pharyngo-maxillary inflammation without abscess	.....	Cervical adenitis without abscess	.....	.....	.....	None	Spontaneous subsidence Subsequent tonsillectomy	Recovery
No. 73 Emily S.	43 yrs. F.	Dental infection following extraction	.....	Submaxillary abscess with marked cellulitis (Ludwig's)	.....	.....	.....	.....	None	External drainage	Recovery

Cases	Age and Sex	Etiology Portal of Entrance	REGIONAL PATHOLOGY Pharyngo-maxillary Space	Sub-maxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral Bucco-phayngeal Pretracheal	Carotid Sheath	Complications	Treatment Received	Result
No. 74 Henry Z.	50 yrs. M.	Dental infection following extraction	.....	Submax-illary infection	.....	.....	.....	.....	None Diabetes	Spontan-eous subsi-dence	Recovery
No. 75 Lewis P.	15 yrs. M.	Pharynx Tonsils out	.....	.....	Cervical gland abscess Deep chain	.....	.....	.....	Severe acute glomerular nephritis	External drainage	Recovery
No. 76 Robert R.	7 yrs. M.	Tonsils	.....	.....	Cervical gland abscess Deep chain	.....	.....	.....	Acute otitis media, supp. opposite side	—	Recovery
No. 77 Donald L.	4 yrs. M.	Tonsils and pharynx	Pharyngo-maxillary abscess	.....	Cervical adenitis, secondary	.....	.....	.....	None	Per-oral incision	Recovery
No. 78 Robert K.	12 yrs. M.	Tonsil	Pharynx-maxillary abscess	.....	Cervical adenitis, secondary	.....	.....	.....	None	Per-oral incision after tonsillec-tomy	Recovery

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LXXVI

CEREBRAL EDEMA AS A CAUSE OF INTRACRANIAL HYPERTENSION OF OTITIC ORIGIN*

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The syndrome of increased intracranial pressure occurring during the course of middle ear suppuration, but not associated with focal or diffuse intracranial inflammation, has been emphasized by otologists and neurologists only in comparatively recent years. It consists of the signs and symptoms of intracranial hypertension, namely: headache, vomiting and papilledema, the absence of positive focal neurological findings and the finding of normal cerebrospinal fluid under increased pressure. An initial diagnosis of brain abscess is usually made but is finally abandoned because of the absence of focal symptomatology and because clinical recovery occurs following single or repeated lumbar punctures or following negative exploratory craniotomy with palliative decompression. It has been generally thought that the intracranial hypertension is due to some disturbance in the dynamics of the intracranial cerebrospinal fluid, i. e., a disturbance in the normal equilibrium between the productive and absorptive mechanisms. Any lesion which obstructs the normal circulation of cerebrospinal fluid will produce a communicating or noncommunicating hydrocephalus, depending upon the anatomical site of obstruction. Following otitis and mastoiditis, localized adhesive leptomeningeal processes may cause a damming back of fluid and a dilatation of the ventricular system by blocking the foramina of Magendie and Luschka in the posterior fossa or by obstructing the flow in the middle fossa or by pressure from an encysted collection of subarachnoid fluid in the posterior fossa. These cases of chronic adhesive arachnoiditis should be carefully differentiated from the cases of intracranial hypertension under discussion, since the pathology, course and treatment are usually quite different. Unfortunately, this distinction is not always made in the literature on the subject.

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Passot[1] first clearly defined the clinical syndrome of otogenous intracranial hypertension not due to intracranial inflammatory disease in 1913, and gave it the name "meningeal hydrops," on the assumption of an excessive amount of fluid in the meninges. The syndrome was little known, however, until Symonds[2] emphasized it by reporting three cases in 1931. A small number of cases have since been reported, with reviews of the literature, especially recently by Williams.[3] Symonds rejected the idea of a "serous meningitis" and Passot's conception of "meningeal hydrops." He considered that a communicating hydrocephalus existed in these cases, due either to an excessive production or a defective absorption of cerebrospinal fluid, and proposed the term "otitic hydrocephalus." He found a dilated lateral ventricle on ventricular puncture in one case but no ventricular punctures or air studies were done in the other two.

In 1937, Symonds[4] reported five more cases, all of which recovered with lumbar punctures, in whom no air studies were done, so that the assumption of a communicating hydrocephalus was not proven. McAlpine,[5] in a paper entitled "Toxic Hydrocephalus," reported five cases associated with other than otitic infections. These patients had the typical clinical picture and all recovered with lumbar punctures, intravenous hypertonic saline and removal of the focus of infection. The author considered the "hydrocephalus" to be external in these cases, i. e., an excessive amount of fluid in the subarachnoid spaces, but no exploratory craniotomies, ventricular estimations or pneumographic studies were carried out to substantiate this hypothesis. He postulated, as did Symonds, an overproduction of fluid, possibly due to a functional disturbance of the choroid plexus or defective absorption, or a combination of these two mechanisms, due to some toxic or possibly allergic reaction to the focus of infection. McConnell,[6] in his discussion of abnormalities in the amount and circulation of cerebrospinal fluid associated with otitis media, mentions communicating hydrocephalus with an excess of fluid as one type. His six reported cases, however, are of the obstructive type due to encysted collections of fluid in the subarachnoid space.

Under the title of "Otitic Hydrocephalus," Williams[3a] reports four cases, the last two of which are of the obstructive adhesive arachnoiditis variety. In his first case, however, a large quantity of fluid was found in the subarachnoid space when subtemporal decompression was carried out. After the withdrawal of 70 cc. of fluid from the temporal horn of the lateral ventricle, air was

injected. In the roentgenograms air was seen over the cortex but none in the ventricles. This finding is difficult to reconcile with the evidences of a dilated ventricle. In the second case a collection of fluid was found in the subarachnoid space at subtemporal decompression. No description of the condition of the brain itself is given. Williams concludes that "otitic hydrocephalus" is a type of acute or subacute hydrocephalus produced by bacteria or toxins, and that it is so frequently associated with otitis because of the frequency of involvement of the dura in middle ear suppuration.

These cases of so-called otitic hydrocephalus selected from the literature are presented because I was impressed, first, by the pneumographic and operative findings in the case which forms the basis of this communication and, second, by the absence of definite evidence of dilatation of the ventricles in most of the cases reported in the literature. This point has been emphasized recently by Davidoff and Dyke[7] in a report of fifteen cases of intracranial hypertension with papilledema and without objective neurological findings. Their cases correspond in every way with those of so-called otitic hydrocephalus, except that otitis was present at the onset in only four. Ventriculography was performed in all, revealing normal or smaller than normal ventricles. Subtemporal decompression was done in all fifteen cases. In each instance the subarachnoid spaces were distended with fluid and the underlying brain was found to be edematous. Recovery occurred in all but one patient, who died three months later of another cause. The brain in this case showed evidences of cerebral edema which, in the opinion of the authors, may have been due to poor fixation. In the fourteen recovered cases the increased intracranial pressure slowly subsided over a period of months or years.

Davidoff and Dyke object to Symond's term "otitic hydrocephalus" because the majority of cases are not associated with otitis media and air studies show no hydrocephalus. Instead they use the term "serous meningitis" to indicate the meningeal site of the excess fluid. In a subsequent communication[8] they go back to Passot's original term and suggest the name "hypertensive meningeal hydrops." This concept of an excessive amount of fluid in the subarachnoid spaces fails, however, to explain adequately the ventriculographic and operative findings of small ventricles, as one would expect to find a gradual dilatation of the ventricular system and the production of a noncommunicating hydrocephalus in a condition in which the amount of ventriculo-subarachnoid fluid progressively increased to the point of clinical hypertension. The authors

describe but do not emphasize the findings of gross edema of the brain tissue itself in all their cases and histological evidence of edema in the one autopsied case. It seems reasonable to assume that the small size of the ventricles in these cases is due to the cerebral edema, which is probably responsible, in part at any rate, for the intracranial hypertension. It is questionable, therefore, whether the name hypertensive meningeal hydrops takes into adequate account the role played by the cerebral edema in the production of the hypertensive syndrome in these cases.

This type of pathology was found by Dandy[9] to have produced marked intracranial hypertension in a series of twenty-two cases simulating brain tumor. These patients had signs and symptoms of increased intracranial pressure, various neurological symptoms and practically negative neurological findings. The cerebrospinal fluid was always normal except for increased pressure varying from 250 to 550 mm. of water. Ventriculography uniformly revealed small and, in some instances, undersized symmetrical ventricles in all cases, and at operation the brain was always found to be edematous and to bulge markedly. All patients recovered; the papilledema subsided but it often took months or years before the pressure, indicated by the amount of bulging of the decompression, entirely subsided. In his concluding remarks Dandy emphasizes the point that the increased intracranial pressure in these cases is due to an abnormal amount of fluid in the brain itself, not in the meninges. He is unable to offer any definite etiological explanation of the disturbance in the dynamics of the intracranial fluid, but suggests that it might be due to changes in the cerebral vascular bed, as he noted rapid fluctuations in the bulging of the decompressions for months and years after clinical recovery. Sporadic reports have appeared in the literature of similar cases simulating expanding lesions in which the increased intracranial pressure was due to cerebral edema. Clinically these cases present the same syndrome as the cases of so-called otitic hydrocephalus, although they often occur in the absence of any clinical evidence of foci of infection.

The purpose of this communication is the presentation of a case, clinically one of so-called otitic hydrocephalus, but in which air studies and craniotomy failed to reveal any evidence of hydrocephalus, but instead the presence of an intracerebral edema as the cause of the hypertensive syndrome.

REPORT OF A CASE

CASE 1.—The patient was a 10-year-old boy who was admitted to the Michael Reese Hospital on March 26, 1938, with an acute bilateral otitis media. Symptoms and signs of bilateral mastoiditis developed and a bilateral mastoidectomy was done on March 31. At operation, in addition to the bilateral suppurative mastoiditis, pus was found around the left lateral sinus, the wall of which was distinctly whitish. The sinus was exposed for about one inch but was not opened. The right lateral sinus presented a normal appearance. Following the operation the temperature, pulse and respiration returned to normal. During the next week the boy complained of headaches and he vomited on several occasions. On April 7 the ophthalmologist reported normal fundi and the neurological examination revealed no objective signs of intracranial disease. On April 14, beginning papilledema and minute retinal hemorrhages were noted. The papilledema then increased progressively but there were no further complaints of headache or vomiting. Neurological examination on April 27 was again essentially negative except for the papilledema. Lumbar puncture at this time revealed clear fluid under a pressure of 350 mm. of water. The Queckenstedt was normal. There was no increase in cells or total protein. A tentative diagnosis of otitic hydrocephalus was made because of the intracranial hypertension and the absence of subjective and objective signs of a focal lesion. The patient had no complaints whatsoever and appeared perfectly well except for a low-grade fever. Lumbar puncture was repeated on April 26 and again April 29, the pressure being 200 and 180 mm. of water, respectively. Coincident with the decrease in intracranial hypertension the papilledema began to subside so that on May 6 both optic disks were practically flat. Visual fields at this time showed a moderate concentric constriction. The mastoid wounds were healing normally and it was felt that the intracranial complication had subsided. On May 19, however, papilledema of two diopters was again noted. There were no subjective complaints of headache or vomiting and neurological examination was completely negative. Lumbar puncture revealed clear fluid under increased pressure, with normal cell count. Repeated punctures were then done every other day, with removal of about 30 cc. of fluid each time. The pressures continued to be elevated, varying usually between 280 and 430 mm. On one occasion the pressure was 600. The ajala indices, at first between 7.2 and 7.5, dropped to 5.2 to 5.4. Magnesium sulphate by mouth and intravenous hypertonic dextrose were used with no sustained reduction in the spinal fluid pressure. Despite these various measures the papilledema gradually increased, some reduction in visual acuity developed and the constriction of the visual fields became more marked. The patient ran an intermittent low-grade fever between 99 and 101 degrees Fahrenheit. There was no leucocytosis. The mastoid wounds had healed, the ears were dry and there was no evidence of infection in the paranasal sinuses. The Tobey-Ayer test was repeatedly negative, indicating that both lateral sinuses were patent.

At this point the nature of the intracranial disturbance was distinctly unclear. The usual case of so-called otitic hydrocephalus responds promptly to drainage of the cerebrospinal fluid. The persistently increased pressure therefore indicated the probability of some other lesion. A localized leptomeningeal process in the middle fossa would obstruct the flow of fluid, interefere with proper absorption and produce a damming back of fluid and a communicating hydrocephalus. The high pressures and the free flow of fluid by lumbar route indicated that there was no block in the posterior fossa. The other possibility would

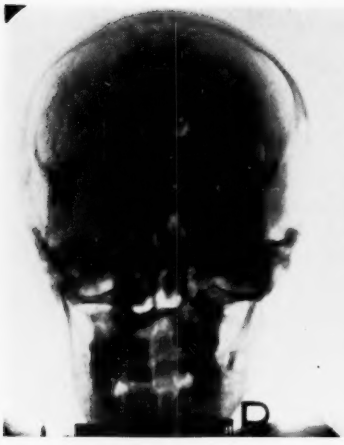

Fig. 1. Ventriculogram.

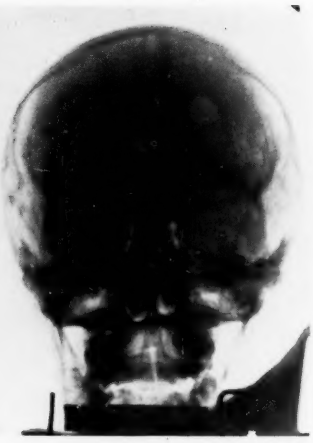

Fig. 2. Encephalogram.

be an expanding lesion such as an intracerebral abscess, which produced no signs or symptoms except increased pressure. To investigate these possibilities, ventriculography was performed and revealed small, symmetrical ventricles (Fig. 1). There was no evidence of any expanding lesion nor of a hydrocephalus. Immediately following the ventriculography the patient developed a left-sided hemiparesis which disappeared in a few days, leaving a very slight residual weakness of the left upper extremity. The spinal fluid pressure remained high, however. The papilledema of three diopters persisted and the reduction in visual acuity and visual field constriction was progressive. In order to prevent any further loss of visual acuity, a palliative subtemporal decompression was done on June 23. At operation a wet, edematous brain which bulged into the cranial defect was found. Following the operation the decompression bulged markedly and was tense, indicating that the intracranial pressure was still increased. No lumbar punctures were done for two weeks, in the expectation that there would be a gradual adjustment in the intracranial pressure. When the decompression continued to bulge and the papilledema persisted unchanged, frequent lumbar punctures with removal of large quantities of fluid were again resorted to. The pressures were still high, varying from 180 to 410 mm. of water. Immediately following spinal drainage the decompression would become flat but would resume the bulging within several hours. Intravenous hypertonic sucrose given at frequent intervals and magnesium sulphate by mouth and intramuscularly in a 25 per cent solution produced only temporary or no reduction in pressure. One month after the decompression the visual acuity was 0.4 plus 1/0.5 plus 2, and the papilledema was still about three diopters. On July 25, therefore, endolumbar encephalography was done, disclosing a normal sized, symmetrical ventricular system (Fig. 2). There was no evidence of an expanding lesion nor of dilated ventricles or dilated subarachnoid spaces. About six weeks after the decompression the retinal hemorrhages began to absorb and the papilledema showed signs of sub-

siding. Visual acuity improved and the visual fields became larger. The decompression continued to bulge as before and the last spinal fluid pressure reading done on August 17 was 280 mm. of water. During all this time the patient had no subjective complaints whatsoever. He was up and about the ward, and physical activity seemed to have no harmful effect on him, so that he was discharged from the hospital on August 24, 1938. The patient has been followed at regular intervals in the outpatient department and there have been no further symptoms. Both optic disks remain flat; visual acuity and visual fields are essentially normal. The decompression continues to bulge considerably. It is of interest that the degree of bulging is variable, increasing during emotional excitement and physical exertion and decreasing during periods of rest and relaxation.

DISCUSSION

This case is presented not only because of the paucity of similar reports in the literature but because it demonstrates a type of pathology of otogenous intracranial hypertension, so-called otitic hydrocephalus, which has received little or no attention in most discussions of the subject. Following Symonds'[2] conception of otitic hydrocephalus, most writers have assumed that the excessive collections of fluid were in the meninges and ventricles, but in most cases no evidence is presented to confirm this assumption. The findings of intracerebral edema, small ventricles and undistended subarachnoid spaces indicate, along with the cases of Dandy[9] and Davidoff and Dyke,[7, 8] that the postulation of a hydrocephalus, i. e., dilated ventricles, is incorrect in many cases. The natural query is whether or not this condition of intracerebral edema is a frequent or only a rare cause of intracranial hypertension. It is impossible to answer this question satisfactorily at the present time for the simple reason that pneumographic studies and craniotomy findings have been reported in relatively few cases. Most cases recover with simple lumbar drainage of fluid, so that the type of intracranial disturbance is never known. One wonders in how many of these cases there may have been a transitory cerebral edema. Such a case was recently reported by Ferey,[10] who found an acute cerebral edema which subsided after exploratory cranitomy and tapping of the small undilated ventricles in a patient who developed acute cerebral symptoms during a suppurative mastoiditis. In my case the hypertensive syndrome, including the papilledema, completely subsided in the course of three weeks with three lumbar punctures. It was naturally assumed that we were dealing with what is usually considered to be an otitic hydrocephalus. Had the hypertensive symptom not recurred we would never had corrected our erroneous impression. Why the hypertensive condition recurred and why it failed to subside following lumbar drainage the second time is completely unclear.

The pathophysiology and etiology of this intracerebral edema is as little known as is its frequency. Symonds[4] has recently advanced the hypothesis that so-called otitic hydrocephalus may be due to partial thrombosis of the posterior longitudinal sinus, resulting in an interference with normal absorption of cerebrospinal fluid through the arachnoidal villi. This partial thrombosis, which would then undergo subsequent organization and recanalization, he ascribes to infection spreading from a lateral sinus.

That even partial occlusion of the posterior longitudinal sinus would lead to the type of hydrocephalus postulated by Symonds seems rather doubtful. In thrombosis of this sinus one usually finds profound disturbances which result from the obstruction of the venous flow into the sinus, namely, venous engorgement and congestion, cerebral edema, extravasation of blood into the subarachnoid space and into the brain substance and frequently necrosis of brain tissue. When dilatation of the ventricles occurs, it is secondary to some other pathology, such as organization of blood in the subarachnoid space, atrophy of cerebral tissue, etc. Study of the original reports of the cases of Bailey and Hass, which were used by Symonds to support his thesis, confirms this impression. Their first case had blood in the cerebrospinal fluid during life and at autopsy yellow gelatinous fluid was found over the entire convexity in addition to hemorrhages and softening in the brain. The hypertensive syndrome in this case must have been due to the cerebral edema and hemorrhage. In the second case internal and external hydrocephalus was present but there were bilateral subdural hematomata due to the longitudinal sinus thrombosis and the resulting venous stasis. The third case had an old hemorrhagic leptomeningeal process due to the subarachnoid bleeding following the longitudinal sinus thrombosis, and necrosis and atrophy of brain tissue, so that the internal hydrocephalus was a result of a combination of lesions. A case of Ellis'[12] of hydrocephalus following longitudinal sinus thrombosis, quoted by Symonds, had also had subarachnoid bleeding which was probably responsible for the interference with the cerebrospinal fluid circulation and the ventricular dilatation. A most interesting case of longitudinal sinus thrombosis with recovery was reported by Frenckner.[13] During a chronic suppurative otitis, the patient developed a typical intracranial hypertensive syndrome. Ventriculography revealed normal ventricles. By means of longitudinal sinus puncture, which failed to find blood and internal carotid arteriophlebography, the author was able to make a positive diagnosis of longitudinal sinus thrombosis. Symonds uses this case as an example of "otitic hydrocephalus," but the finding of normal ventricles

leaves no doubt that the intracranial hypertension must have been produced by some other mechanism, most probably cerebral edema and possibly hemorrhage due to the venous stasis and congestion. A normal ventriculogram was also reported by Doyle[14] in a fatal case of longitudinal sinus thrombosis, which had had, during life, signs and symptoms of increased intracranial pressure. These cases indicate that posterior longitudinal sinus thrombosis may rarely be responsible for the hypertensive syndrome, but its infrequent occurence and the usually severe symptomatology argue against its being of frequent etiological importance.

It has long been known, especially to otologists, that obstruction of the lateral sinus or the jugular vein not infrequently produces increased intracranial pressure and papilledema. In these cases the intracranial hypertension is evidently related to the interference with the venous return from the intracranial contents, resulting apparently in venous stasis and focal or diffuse cerebral edema. In discussing this subject, Nielsen and Courville[15] emphasize the role played by the lack of valves, the tortuosity and the negative pressure in the venous sinuses in producing, under suitable conditions, a slowing and even a reversal of circulation. Many authors have pointed out the anatomical variations in the lateral sinuses and mastoid veins, especially the difference in size between the two sinuses in some individuals. Ersner and Myers[16] especially have suggested that the venous circulatory disturbances associated with suppuration of the temporal bone are of etiological importance in cases of so-called otitic hydrocephalus. These authors consider the variability in size of the venous channels of great importance, reasoning that vasostasis resulting from temporal bone pathology will result, in the presence of narrow venous channels, in intracranial venous stasis and hypertension. In reviewing cases of otitic hydrocephalus, Symonds[4] was struck by the high incidence of lateral sinus infection. In my case there was a definite perisinus abscess and the alterations of the appearance of the sinus suggested an infection of the wall itself, but no obstruction as far as can be ascertained by the negative Tobey-Ayer test. Ersner and Myers[16] point out that venous stasis and increased intracranial pressure may occur in the absence of thrombosis of the sinus itself, so that the absence of such obstruction in my case does not exclude the possibility of venous stasis. Partial obstruction by mural thrombi might result in sufficient slowing of the venous circulation to produce congestion, edema and intracranial hypertension, which would subsequently disappear as an adequate venous flow was established through the same or other channels. The duration of the venous stasis and intracranial hyper-

tension would depend not only upon the degree of occlusion but also upon the size of the venous channels available for establishing collateral circulation. It has been well established that changes in venous pressure, either generalized or confined to the head, are accompanied by similar changes in the cerebrospinal fluid pressure (Merritt and Fremont-Smith[17]). Interference with the intracranial flow due to partial thrombosis of a lateral sinus and its tributaries resulting in venous stasis would increase the intracranial venous pressure and, concomitantly, the cerebrospinal fluid pressure. The actual mechanism responsible for this relationship between the intracranial venous and cerebrospinal fluid pressures requires much clarification, as does the problem of cerebrospinal fluid formation and absorption, and is beyond the scope of this report. The fluctuations in the degree of bulging of the decompression with exertion or excitement is suggestive, as pointed out by Dandy in his cases, that changes in the cerebral vascular bed directly influence the intracranial cerebrospinal fluid pressure.

SUMMARY AND CONCLUSION

A case of otogenous intracranial hypertension is described in which the pneumographic and operative findings indicated that the increased pressure was due to an excessive amount of fluid in the brain itself rather than, as is generally assumed, in the ventricles and subarachnoid spaces. Similar cases, with and without foci of suppuration in the temporal bone, have been described in the literature and have been variously termed serous meningitis, hypertensive meningeal hydrops, and rarely cerebral edema. It is suggested that the commonly accepted concept of otitic hydrocephalus is probably incorrect in many cases to which the term is applied, as the presence of dilated subarachnoid channels and a communicating hydrocephalus has not been demonstrated in most of the reported cases. The term "otitic hydrocephalus" is used in an extremly loose and vague fashion both in the otologic and neurologic literature, and often includes cases of obstructive noncommunicating hydrocephalus due to arachnoidal adhesions, encysted subarachnoid collections of fluid and reactive meningitis, as well as cases of communicating hydrocephalus. It seems to the writer that careful efforts should be made to determine more accurately what type of intracranial disturbance is present. In those cases in which the hypertensive syndrome subsides after simple lumbar drainage, it seems best to apply simply the descriptive term "hypertensive syndrome" rather than such terms as "otitic hydrocephalus" or "hypertensive meningeal hydrops," for the reasons already mentioned. In cases which

do not respond to lumbar drainage and dehydrating measures, pneumographic studies (preferably ventriculography) should be done. In such cases the nature of the pathology will then be at least partially disclosed and the proper therapeutic procedures can be carried out.

In the case under discussion, a persistent cerebral edema was found to be the cause of the intracranial hypertension and the suggestion is offered that venous stasis due to infection in the dural sinuses might be the underlying etiology. It is also suggested that cerebral edema might be a much more frequent cause of the otogenous hypertensive syndrome than is generally supposed.

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LXXVII

IRRIGATION OF THE MAXILLARY SINUS THROUGH THE OSTIUM: A CLINICAL STUDY OF FIVE HUNDRED CASES

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The accessibility of the natural ostium of the maxillary sinus has been a controversial point for many years, but, during the past decade, more information has appeared in the literature to indicate that the treatment of maxillary sinusitis by this route is receiving increasing attention. The greater number of contributions, however, regarding the accessibility of the natural ostium, have been made by anatomists, and comparatively little has been written from a clinical standpoint.

According to Schaeffer,[8] the situation of the maxillary ostium of the adult corresponds to that of the primitive maxillary pouch. The pouch gradually develops into the pyramid shaped cavity of adult, leaving the place of communication with the infundibulum ethmoidale at the point of primary evagination. The maxillary sinus, therefore, communicates directly with the infundibulum ethmoidale. The infundibulum is a groove or gutter situated upon the lateral nasal wall, and "it is bounded cephalically by the caudal surface of the bulla ethmoidalis throughout the greater part of its extent, save ventrally and superiorly, where the bulla is replaced by some anterior ethmoidal cells: the caudal and mesial boundary of the groove is formed by the lateral surface of the processus uncinatus."

The infundibulum communicates with the middle meatus through the hiatus semilunaris, which is a narrow slit or semilunar cleft between the bulla and a well defined grooved processus uncinatus of the ethmoid bone. This semilunar hiatus is an important opening, since it is a communication between the middle meatus and the gutter-like infundibulum ethmoidale. The size of the bulla ethmoidalis greatly influences the dimensions of the hiatus semilunaris and thereby influences the accessibility of the ostium of the maxillary sinus. Hence, in approaching the maxillary sinus through the middle meatus, a series of openings must be traversed, namely, the hiatus semilunaris, into the infundibulum, through the maxil-

lary ostium, into the sinus. The maxillary ostium, as a rule, is situated in the most dependent part of the infundibulum and varies from 1 to 12 mm. in distance from the hiatus semilunaris. The distance is dependent upon the width of the processus uncinatus. The maxillary ostium may be round, but as a rule, is either oval or elliptical. Schaeffer gives its dimensions as varying from 1 to 22 mm. in length and from 1 to 6 mm. in width. The accessory maxillary ostium is usually situated in the membranous portions of the lateral wall of the middle meatus and at about the junction of the middle and posterior third of the inferior turbinated bone.

How frequently the accessory ostium is present and the reasons for its presence are still controversial subjects. The accessory ostium, when present, furnishes a direct communication between the maxillary sinus and the middle meatus, and can be used for the purpose of irrigation of the antrum.

Opinions as to the accessibility of the natural ostium vary in percentages from zero to as high as 85 per cent or 90 per cent. These opinions usually are based upon study of the cadaver and, while this method of investigation yields exceedingly valuable information, it can be greatly augmented by routine practice on the living subject.

Schaeffer[8] says that an examination of a large series of specimens leads him to believe that it is impossible clinically, in the majority of cases, to sound the maxillary sinus through its normal ostium. Mosher[3] concurs in this opinion, and says that it is difficult, if not impossible, in the majority of cases to catheterize the ostium of the antrum. From the surgical standpoint, as he points out, this makes but very little difference because it is easy to break into the antrum near the ostium through the membranous area in which the ostium is placed.

A study of 163 specimens, made by Van Alyea,[12] showed that the ostium or an accessory opening was easily accessible in eighty-nine, or 54.5 per cent. Thirty-two per cent could be entered with difficulty, while the remaining 13.5 per cent were entirely inaccessible. The factors which prevented entrance of the ostium in his series included a narrow hiatus semilunaris, overhanging bulla, a high uncinate wall, deviated septums, and cystic or bulky hypertrophy of the area of the middle turbinated bone. Van Alyea noted the following factors as aiding cannulization: atrophy, low uncinate walls, large ostia, accessory ostia, and ostia situated at the posterior tip of the infundibulum. The incidence of accessory ostia in his series was 25 per cent.

Myerson[6] reported favorable results in living subjects, and stated that he had used the natural ostium for irrigation of the maxillary sinus in 81 per cent of his patients. Futch reported a clinical experience in 1200 cases and said that it had been possible to irrigate the maxillary sinus through the middle meatus without puncture in 72 per cent. Approximately one-third of these were irrigated by way of an accessory ostium.

The idea of using the natural ostium to irrigate the maxillary sinus has met with considerable opposition every time the subject has been presented. Many rhinologists have rather half-heartedly tried to probe the natural ostium, and if this proved at all difficult, they have abandoned the procedure and have resorted to the old method of irrigation by inferior meatal puncture. Others routinely use the natural or accessory ostium for irrigation in cases of maxillary sinusitis. These discrepancies and controversies have at least stimulated considerable interest in the matter, and some rhinologists maintain that the advantages of this procedure far outweigh the objections that have been raised against it.

Futch has summarized the advantages of this procedure very aptly: "(1) Danger of air embolism is reduced; (2) hemorrhage is not experienced; (3) the unpleasantness of pressure is greatly eliminated; (4) infiltration, or emphysema of soft parts is less liable with a dull instrument; (5) nervous patients experience less shock than when a sharp needle is forced through bone, or even membrane; (6) where the ostium is inaccessible, anesthesia is complete for puncture through the middle meatus; (7) shrinking the tissues of the middle meatus, even when puncture has to be performed, has the benefit of an improved aeration and drainage of the sinuses emptying into the middle meatus; (8) a patient who has experienced irrigation through the natural opening will not permit puncture."

Aside from the contention that it is impossible to probe the natural ostium, arguments used against this procedure are that it produces excessive trauma to the mucosa and the adjacent tissue around the natural opening, and that it is impossible to flush the antrum completely when the cannula is in the natural ostium.

Inability to locate the natural ostium should not be used as an argument against using it for irrigation of the maxillary sinus. Continued trial is bound to result in improved technic, and those who have experimented with the procedure report that diligent practice and effort have been rewarded with increasing success. If reasonable care is used, instrumentation should not irritate the mucosa of the surrounding structures. Certainly such instrumentation should not

produce discomfort comparable to that produced by an inferior meatal puncture, with its resultant trauma, discomfort and bleeding. As to the obstruction of the natural ostium by the cannula, the anatomic construction of the ostium, in most instances, allows the insertion of an ordinary small round cannula, without obstruction of the return flow. In my own practice, no difficulty whatever has been experienced in obtaining good return flow with this method of irrigation.

It was when I was associated with the late Dr. William V. Mullin, a decade or more ago, that my interest in irrigation of the maxillary sinus through the natural ostium was aroused. He had been experimenting with the procedure since 1915. Together, we devised a cannula which permits successful irrigation of the antrum through the natural or accessory ostium in nearly all cases.

A series of 500 cases has been studied, in which irrigation of the maxillary sinus through the natural or accessory opening has been carried out, for the relief of purulent maxillary sinusitis. No effort has been made to distinguish between the natural and the accessory ostium in this study, but in all instances, the sinus was irrigated through the middle meatus, without puncture.

This group of cases was unselected, except that those in which the sinus infection was due to existing or previous tooth infection, dental cyst, or maxillary tumor, were excluded. In all instances in this series, infection of the maxillary sinus followed some acute upper respiratory infection. The diagnosis, for the most part, was made by evaluation of the patient's symptoms and finally by recovery of purulent material by irrigation through the natural ostium. Maxillary sinusitus probably exists much more frequently than is indicated by symptoms, for it has been possible, in many instances, to recover purulent material from the maxillary sinus when no symptoms referable to the antrum were elicited. In many cases, prolonged postnasal discharge following an upper respiratory infection, or mild, persistent head cold with so-called stuffy nose and a full feeling, were immediately relieved by a few irrigations of the maxillary sinus.

Two hundred and twenty-four of these patients were males; 276 were females. The greatest number (65.4 per cent) were between twenty and forty years of age. The age distribution for the entire group is shown in Table 1.

The right side was involved in 122 cases and the left side in 119 cases. Both maxillary sinuses were infected in 259 cases. Thus, slightly more than 50 per cent of the patients had bilateral infections. The total number of sinuses treated was 759.

TABLE 1.

AGE DISTRIBUTION

AGE	NUMBER	PER CENT
6 to 10 years	6	1.2
10 to 20 years	59	11.8
20 to 30 years	139	27.8
30 to 40 years	188	37.6
40 to 50 years	70	14.0
50 to 60 years	31	6.2
60 to 70 years	7	1.4

In a study of the symptoms which these patients presented, it was rather surprising to find that acute pain in the maxillary region was not a predominant symptom. It was the chief complaint of only 125 patients, or 25 per cent of the total number. Table 2 gives a rather concise picture of the chief symptoms of which the patients complained upon first examination, and it can be seen that the most common symptom was a mild, persistent head cold and nasal congestion. Many patients of this type are allowed to go on with little or no treatment, and of course, many others do not seek medical attention unless they are suffering discomfort. Nevertheless, those who have had treatment for an infection of the antrum and have obtained relief from their symptoms of stuffy nose and postnasal discharge, by irrigation, are much more likely to seek relief in cases of subsequent infection, even though they are not suffering from definite pain.

In this series of cases, transillumination was a fairly reliable sign of inflammation of the maxillary sinus. Of the 500 patients studied, transillumination was not done in eighty-two cases. In the 418 instances in which it was carried out, the sinuses were transilluminated fairly clearly in only ten cases in which pus was obtained upon irrigation.

In the management of these cases, all irrigations were done through the middle meatus, after an application of 5 per cent cocain solution. The use of epinephrine or any powerful shrinking drug was purposely avoided, because of the tendency to compensatory edema around the ostium, after its application. A blunt-nosed, S-shaped cannula, with two side openings, made of flexible, chrome-plated copper tubing was used. This cannula allows entrance into the maxillary sinus with minimum trauma, because it can be easily

shaped so as to accommodate to almost any type of deformity of the septum. No attempt was made to distinguish whether natural or accessory ostia were used for entrance into the antrum. With this technic, all the antra were probed successfully without puncture of the middle meatus, and practically all the patients experienced no discomfort following the procedure.

TABLE 2.

SYMPTOMS

SYMPTOM	NUMBER	PER CENT
Maxillary pain	125	25.0
Headache, frontal and orbital	55	11.0
Purulent nasal discharge	108	21.6
Postnasal discharge	29	5.8
Mild head cold and nasal congestion	140	28.0
Cough	12	2.4
Chronic history	28	5.6
Temperature elevated	1	.2
Edema of the eye	1	.2
Impaired vision	1	.2

Various solutions have been employed for these irrigations, but the one that is used routinely, and seems to give the least reaction afterwards, is a modified Ringer's solution. This is non-irritating and consequently produces minimum edema of the membrane following irrigation. Some antiseptics, although well diluted, have caused rather severe reactions, and their repeated use was not attempted.

In the few instances in which improvement was not evident after several irrigations, and in which foul, purulent, unorganized secretion persisted, additional study was undertaken by injecting the sinus with lipiodol or some radiopaque oil. If roentgenographic examination revealed sufficient evidence of disease, operation was advised. Even in some cases of this type, however, cure may be effected if irrigations are carried out for a sufficient period of time. One patient in this series, a healthy young man, age eighteen years, had twenty-two irrigations in two months, and has remained completely well over a period of several years. In many cases, however, the

patient prefers to undergo a surgical operation, rather than submit to a prolonged series of treatments.

Thirty-four patients (6.8 per cent) in this series did not respond to irrigation through the natural ostium, and had to be subjected to surgery. In twenty-eight (5.6 per cent) cases, the radical (Caldwell-Luc) type of operation was performed. In six cases (1.2 per cent), an intranasal, inferior meatal window was made. In the latter group, a radical operation was necessary in one case three years after an inferior meatal window, which had failed to provide sufficient drainage, had been made.

In an attempt to arrive at an opinion as to the quantity of treatment required to heal an infected sinus, the total number of irrigations was divided by the total number of antra involved. In the entire group, 759 antra were irrigated 3,247 times. This makes an average of slightly more than four irrigations per antrum. Allowing for individual differences, which depend on various factors, such as the type and severity of the infection, the general condition of the patient, etc., this yields some criterion on which to base the prognosis, in cases of acute infection of the antrum.

In eighty-three of the 500 cases, the patients had a subsequent infection in the sinus. Some of these had more than one infection, so the total number in the series was 115 subsequent infections. In every instance, these had to be regarded as subsequent infections, rather than recurrences, for the patients had been previously discharged as cured (Table 3).

TABLE 3.

SINUS INFECTIONS SUBSEQUENT TO TREATMENT
(83 PATIENTS)

TIME ELAPSED SINCE TREATMENT	NUMBER
Less than 1 year	38
1 year	16
2 years	14
3 years	17
4 years	8
5 years	6
6 years	9
7 years	4
8 years	3
Total	115

SUMMARY

The accessibility of the natural ostium for irrigation of the maxillary sinus has been the subject of numerous anatomic and clinical studies, which have resulted in widely divergent opinions from various sources. Some rhinologists regard irrigation by this method as impracticable, if not impossible, while others consider it the procedure of preference and report successful results in 85 to 90 per cent of cases.

My own experience corroborates the opinion of the latter group, for, with the use of a special cannula made of flexible chrome-plated copper tubing which can be accommodated to various types of nasal deformities, it has been possible to probe the natural ostium for irrigation of the antrum in all but a few exceptional cases.

A series of five hundred cases of maxillary sinusitis has been reviewed. In all instances, the maxillary sinus was irrigated through the natural or accessory ostium. In the group there were 224 males and 276 females, of whom 65.4 per cent were between twenty and forty years of age. In slightly more than half the patients, the sinus infection was bilateral. Acute pain was the chief complaint in only one-fourth of the cases; the most common symptom was mild, persistent nasal congestion. The average number of irrigations of each infected sinus in this series was four. In only 6.8 per cent was operation necessary. Subsequent infections occurred in only eighty-three of the five hundred patients.

Irrigation of the antrum through the natural or accessory opening is preferable to inferior meatal puncture, because it causes less inflammation and trauma and minimizes the danger of hemorrhage, and because it causes much less shock and discomfort to the patient both during and after the treatment.

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LXXVIII

TESTING OF THE TRANSMISSION APPARATUS WITH THE BONE-CONDUCTION RECEIVER

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TECHNIC AND METHOD OF THE TESTS

The cochlea is situated rather deep below the surface of the skull. The acoustic vibrations have to be very strong in order to reach the perceptive organ except when they are conveyed to it by a conduction apparatus of pinna, meatus and the structures of the middle ear. For diagnostic purposes we need methods by which the perceptions reaching the cochlea through the bone can be separated from those conducted along the special channel. The healthy ear hears the threshold intensities of a tuning fork stem placed against the mastoid process predominantly through the skull and the threshold intensities of the prongs placed into the proximity of the meatal opening exclusively through the conduction apparatus. If the ear is diseased, the two thresholds may approach and the separation of the two kinds of stimuli becomes difficult. Besides that, for finer, and especially for quantitative, diagnostic conclusions, it is of paramount importance that both thresholds should be elicited by the same source of stimulus. This postulate is not fulfilled by the tuning fork. Its stem and its prongs are two different instruments, each having a different initial intensity and a different damping constant.

The audiometer eliminates the difference in damping constants but again has two different sources of stimulus, namely, the air conduction receiver and the bone conduction receiver. The error which is produced by comparing the thresholds obtained from the two receivers could be subtracted if we knew the intensities of both sources of sound. The energetic data of the resonant membrane in the air-conduction receiver are based on Dr. Fletcher's "normal" and are known. But the energetic constants of the end of the bone conduction receiver resting on the skin behind the ear are difficult to establish. Besides that the defective ear listening to the air conduction receiver registers hardly more than telephone hearing because the higher intensities penetrate the insulation of the receiver and are

transfered by direct contact to the tissues of the pinna and of the skull. That causes the well-known bone conduction error of the air conduction receiver, which is larger than the bone conduction error of loudly sounding prongs before the obstructed ear.

For such reasons the author began to test experimentally both the perception organ and the conduction mechanism with one of the receivers only. The air conduction receiver was not considered appropriate. When it is held behind the ear too much sound escapes from it and is perceived through the meatus. Therefore the bone conduction receiver No. D 80904 of the 2B Western Electric Audiometer was used for the tests. The amount of sound escaping from this instrument was measured on the ears of a young observer whose air-conduction and bone-conduction thresholds approximated very closely the normals of the audiometer. The receiver was held in the usual position behind the ear but not in contact with the mastoid process. The tones heard under such arrangement are represented in Fig. 1 by curves marked 1aEr and 1aEl. If the homolateral ear was closed with a finger, practically the same curves were obtained, evidently from the spreading of the escaped sounds to the opposite ear. When the hearing tests were performed on other ears the bone-conduction curves were checked for escape whenever their values approximated the values of the escape curves 1aEr and 1aEl. But the escape only rarely equalled or exceeded the bone-conduction threshold.

Two methods were considered for testing of the transmission apparatus by the bone-conduction receiver. The one would imitate the tuning fork test for air conduction by holding the end of the receiver close to the meatus. But then the same errors and difficulties would arise as in the tuning fork tests: The distance from the meatus would vary and the metal would occasionally touch the tragus or the pinna. Therefore the end of the receiver was placed against the tragus and this cartilaginous organ was forced into the meatal orifice. Naturally such arrangement rendered the test different from the routine air-conduction tests. The vibrations of the instrument were primarily transmitted by direct contact to the cartilaginous and cutaneous tissues around the meatal opening. From there they propagated through the air enclosed in the meatus and through the soft and the bony tissues of the skull. The closed cavity of the meatus inevitably resembled a sounding box in which the impact of sound waves against the tympanic membrane was reinforced by the reflected portion of the waves. So much had to be expected theoretically before the experimentation began. It re-

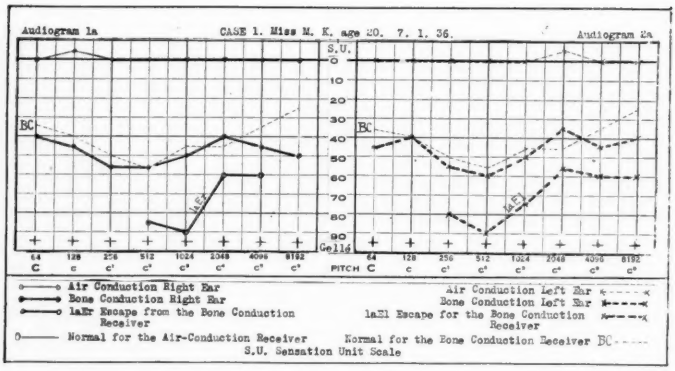

Fig. 1. Air conduction and bone conduction of an observer whose thresholds almost coincide with the normals of the 2B Western Electric Audiometer. Curves 1aEr and 1aEl are records of "escape" from the bone-conduction receiver No. D 80904.

mained to analyze the result of such hearing tests and to evaluate their character, meaning and usefulness.

All experiments were performed in the course of routine office work as a part of general examination of the hearing organs. The history was noted, otoscopic and nose examination were performed. The nasopharynx was inspected with Beck's nasopharyngoscope. Patency of the eustachian tubes was examined after the hearing tests. Thus the influence of inflation on the hearing threshold was eliminated. Inflation of the tubes was performed without the use of cocain or epinephrine, because their application changes the lumen of the orifice and of the tubes.

The hearing tests were made in a uniform order: (1) Air-conduction test of the right ear. (2) Air-conduction test of the left ear. (3) Bone-conduction test of the right ear. (4) Test with bone-conduction receiver placed over the impacted right tragus. (This test shall be called shortly "the tragus test.") (5) Bone-conduction test of the left ear. (6) Tragus test of the left ear. (7) The Weber test. (The Weber test will not be discussed in the present study.) The seven tests were usually taken in one session lasting 20 to 30 minutes. After the patient had rested a while or at the next visit the Gellé test was performed first on the

better, then the worse ear. The tubes were inflated after the Gellé test. In some cases the examination was supplementd by masking, Bing test, paracousis test, etc. All unnecessary therapy was avoided until all the tests were recorded. During the course of treatment the patients were retested as often as the pathology and the experimental interest required and as often as the time and the patient's psychology allowed.

Barany's modification of the Gellé test was accepted for all experiments. In this modification we used a T-shaped auscultation tubes, two ends of which have ear pieces well coated with vaseline, the third a mouthpiece. The former are placed in one ear of the patient and of the examiner and the latter is held in the examiner's mouth. The bone-conduction receiver was originally placed on the metal T-joint of the tube as Barany advised. But later it was found that some tones can be better forced into the cochlea by placing the receiver behind the ear, over the tragus or over the teeth. The Barany modification is technically less difficult if the stapes is moved by suction instead of blowing into the ear. The response of the healthy ear is the same, viz., the tone sounds weaker no matter whether the pressure in the meatus is decreased or increased. Occasionally an "inverted" Gellé was observed, the sound being intensified by both the positive and the negative changes of the pressure. Inverted responses were recorded by the letter "i". If the patient reported difference in loudness the Gellé was recorded as positive, otherwise it was designated with a minus sign. All tones of the 2B audiometer were used for the Gellé test in each ear. If the audiogram shows no Gellé record in some frequency, it means that the patient could not hear the sound.

The tests were performed in an office which has no outside walls and is located in a residential section. There is about 20 decibels of noise in the room. All tests were started with the 512 double vibration tone and were continued first down, then up the scale. Before the patient signaled the threshold he was thoroughly acquainted with the sound by turning the dial to a sufficiently loud tone. The threshold was then carefully approached from low intensity upward. The results were dictated to the secretary. It was found that the average patient is a good observer under such an arrangement. Only three audiograms in a series of about 100 patients had to be rejected because the patient was distracted. The tests were recorded by pencils of different colors: air conduction by a lead pencil, bone conduction in red, tragus test in blue, Bing test in green, etc.

THE TRAGUS CURVE OF HEALTHY EARS IN DIFFERENT AGE GROUPS

Figure 2 represents eight healthy ears of persons belonging to seven different age groups.

CASE 1.—M. K., female, aged 20. Audiograms 1 and 2 of Fig. 2, and 1a and 2a of Fig. 1. Only the left ear (audiogram 2) is considered healthy.

On March 5, 1938, the patient reported that two weeks ago she had a cold in the head and ringing in the right ear. The tinnitus lasted only two days and was not associated with vertigo. Two years ago, on June 25, 1936, I had removed impacted cerumen from both her ears. Five weeks later, on July 1, 1936, when the ears appeared perfectly normal, audiograms 1a and 2a of Fig. 1 were obtained. Previous history indicated several attacks of aural pain after summer bathing in childhood. Examination on March 5, 1938, showed a normal right ear, and no pathology in the nose, pharynx and nasopharynx. There was some accumulation of cerumen in the left meatal orifice, which was removed. The epidermis of the left meatus and the drum-head appeared normal.

CASE 2.—M. A., female, age 30. Audiograms 3 and 4. Only the left ear (audiogram 4) is considered healthy.

On December 8, 1937, this patient reported that two days ago she noticed immobility of the right half of her face. In December 1934, Miss M. A. was treated by me for acute purulent antritis of the right side. Examination on December 8, 1937, revealed right Bell's palsy. Otoscopic examination was negative on both sides. There was no acute nasal or nasopharyngeal inflammation. The septum was deflected toward the right side and in contact with the right middle turbinate. Transillumination of the sinuses was negative. Audiograms were taken on December 30, 1937. Inflation of the eustachian tubes on the same date indicated stenosis on the right side. The facial paralysis made an uneventful recovery within several weeks.

CASE 3.—E. C., female, age 46. Audiograms 5 and 6. Only the right ear (audiogram 6) is considered healthy.

On February 7, 1938, the patient complained of noise in both ears, worse on the left side, during the past year. There was no history of dizziness or of aural discharge. The mother was hard of hearing. Otoscopic examination of both ears was negative. Both

middle turbinates were impacted. Nasopharynx was deeply vaulted and congested. Eustachian openings had normal appearance. Inflation on the right side was free, with deep auscultation tone. On the left side the tone was high pitched and the inflation was difficult. Blood pressure was 140/75. Audiometric examination was made on February 7, 1938. The hearing in the left ear improved after several weeks.

CASE 4.—D. S., female, age 54. Audiograms 7 and 8. Only the left ear (audiogram 8) is considered healthy.

Mrs. D. S. reported on September 13, 1938, that her right ear discharged yellowish secretion for three weeks. Similar discharge appeared repeatedly for the past 18 months. The left ear never caused any trouble. Two days ago the patient was seized with an attack of vertigo which lasted three hours and increased with the movement of the head. The history of tinnitus was negative. On examination the meatus of the right ear appeared swollen and was covered with masses of soft, moist epithelium. The right drum-head had a large perforation in the lower and anterior segment. The defect was filled by a granuloma. The left ear showed no pathology except a moderately retracted drum-head. Nose examination revealed a septal spur opposite the left lower turbinate. The breathing space was ample on both sides. There was no inflammatory involvement of the nasal, nasopharyngeal or pharyngeal mucosa. No spontaneous nystagmus could be observed. Eustachian orifices appeared normal. Auscultation of the right tube gave a blowing tone. On the left side the inflation was easy, with a low-pitched tone. The audiograms 7 and 8 were taken on September 16, 1938, with granuloma in the right ear still in situ. The granuloma was later destroyed and cholesteomatous epithelial clumps were removed from the attic.

CASE 5.—L. W., male, age 59. Audiograms 9 and 10. Only the left ear (audiogram 10) is considered healthy.

This patient visited me on May 7, 1938, because of hardness of hearing and wind-like noise in the right ear lasting six months. He suffered from frequent colds but gave no history of dizziness, pain or discharge from the ears. There was accumulated cerumen in the right ear. The wax was removed but on May 11, 1938, the patient still complained of noise in the right ear. It disturbed him especially at night. Otoscopic examination was negative. The nose showed no pathology. The mucosa of the nasopharynx was moderately thickened and covered with thick, scanty mucopus. The

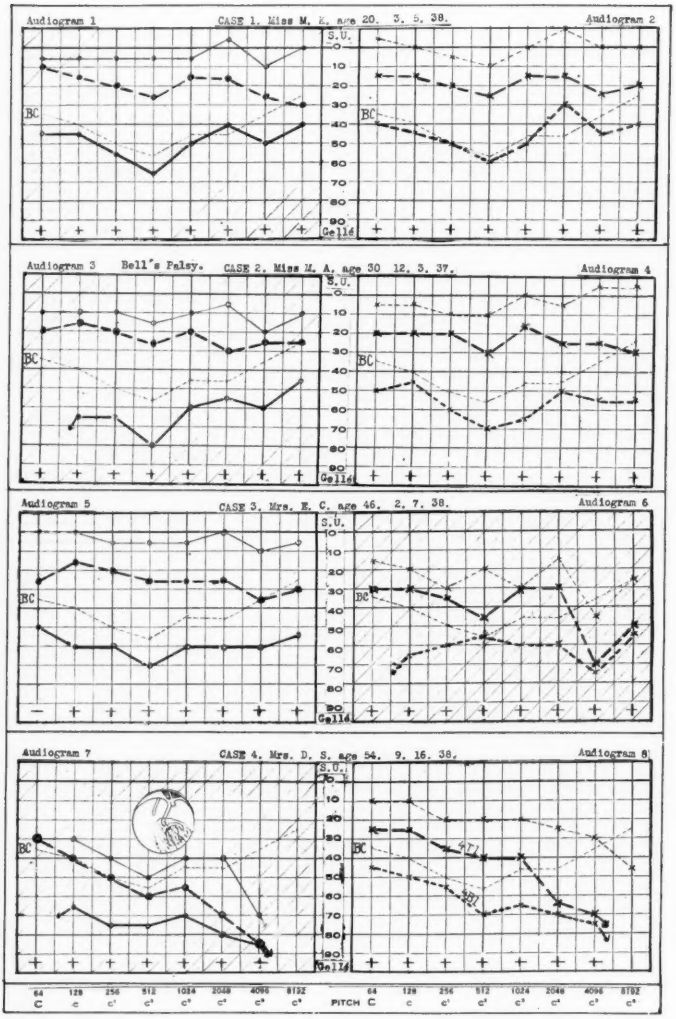
Audiogram 1
CASE 1. Miss M. K. age 20. 3. 5. 38.
Audiogram 2
Audiogram 3
Bell's Palsy.
CASE 2. Miss M. A. age 30 12. 3. 37.
Audiogram 4
Audiogram 5
CASE 3. Mrs. E. C. age 46. 2. 7. 38.
Audiogram 6
Audiogram 7
CASE 4. Mrs. D. S. age 54. 9. 16. 38.
Audiogram 8
S.U.
BC
Gellé
PITCH
64 128 256 512 1024 2048 4096 8192
C c c¹ c² c³ c⁴ c⁵ c⁶

Fig. 2—Part 1.

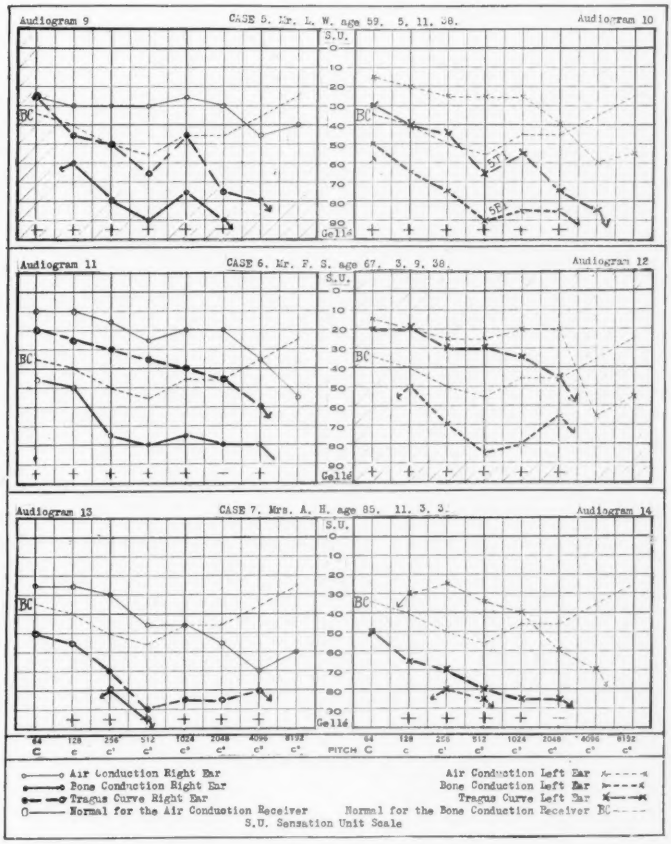

Fig. 2—Part 2.

Fig. 2. Air-conduction, bone-conduction and tragus test in normal ears of persons belonging to different age groups. Audiograms of the pathologic ears are shaded.

eustachian orifices appeared normal. Inflation of the tubes indicated very free passage of the air on both sides. Audiograms were taken on May 11, 1938.

CASE 6.—F. S., male, age 67. Audiograms 11 and 12. Only the right ear (audiogram 11) is considered healthy.

On March 4, 1938, Mr. S. complained of hissing sound in the left ear for the past six months. He was previously under my attention for incipient cataract in both eyes and lipoid deposits in the macula of the left eye. The patient was a heavy smoker. His urine was negative, blood sugar was not increased, blood pressure varied between 180 and 160 systolic and 80 to 85 diastolic. Otoscopic examination was negative. Nasal inspection revealed impacted left middle turbinate with excoriation on the anterior tip. Pharynx appeared normal, mucosa of the nasopharynx was thickened. Eustachian orifices appeared normal, inflation of the tubes indicated no obstruction. The audiograms were taken on March 9, 1938.

CASE 7.—A. H., female, age 85. Audiograms 13 and 14. Both ears are considered healthy.

This patient was examined for hardness of hearing on November 3, 1938. There was no history of ear diseases other than loss of hearing during the past ten or fifteen years. Otoscopy, rhinoscopy and nasopharyngoscopy revealed no pathology. Both eustachian tubes were patent.

Close study of the audiograms and histories of the above selected eight healthy ears may cast some doubt on the absolute absence of lesions in ears Nos. 5, 8 and 10. Ear No. 5 has a history of noises but the audiogram shows very good hearing. In audiograms 8 and 10 the tragus curves (4T1 and 5T1) approach the bone-conduction curves 4B1 and 5B1 in 2048 d.v. and 4096 d.v. This may not be due only to the age, because an older patient of audiogram 11 does not show a similar characteristic. Generally, this group demonstrates the well-known[1, 2] decrease of hearing by both, the bone conduction and the air conduction which begins after the third decade and progresses more rapidly with advancing age and with the pitch.

The tragus curve appears to be conveniently located on the charts between the air-conduction and the bone-conduction curves. It lays nearer the former and perhaps falls toward the latter in the

old age. Mathematical expression of correlation (parallelism) and of the average distances in certain frequency groups might be interesting but it would be of value only if based on large material.

THE TRAGUS CURVE OF GELLÉ NEGATIVE EARS WITH VISIBLE LESIONS

The tragus curves of healthy ears should be compared with such curves in ears with definite, visible and well understood pathology. Six Gellé negative ears were selected for that purpose in Fig. 3. The first of them is represented by audiogram 16. The diagnosis was healed radical mastoidectomy. The second patient (Case No. 9) had chronic purulent otitis media and a simple mastoidectomy on the right side several years ago (Audiogram No. 17). The third patient (Case No. 10) had chronic purulent otitis media with large perforations of both ear drum-heads. The audiograms 19 and 20 were taken when both ears were discharging. The last case (No. 11) had a chronic purulent exacerbating otitis media of both ears. The external auditory meatus of the left ear (audiogram 22) was atretic. The audiograms 21 and 22 were taken during a period when there was no acute exacerbation and no discharge.

CASE 8.—J. K., male, age 36. Audiograms 15 and 16. Mr. J. K. had a radical mastoidectomy on the left side in 1931. It was performed for a chronic discharge lasting since childhood. In 1928 he was injured in a motorcycle accident, after which he remained unconscious for two hours and was subsequently treated for concussion of the brain. For the past nine years he was working daily with a pneumatic hammer. In July, 1937, he complained that the hearing in his right ear becomes worse whenever he works in the noise for long hours. After he rested for several days the hearing recovered. There was no history of deafness in the family, no subjective noises, no vertigo, no abuse of tobacco, quinine or other drugs. The operated left meatal, mastoid and middle ear cavities were well lined with epidermis. The ossicles were absent. The region of fenestrae was covered with a skin graft. Otoscopic examination of the right ear was negative. Nose, nasopharynx, pharynx and eustachian tubes showed no pathology. The audiograms were taken on July 16, 1938, after the patient's hearing began to improve.

CASE 9.—W. O., male, age 15. Audiograms 17 and 18. On November 2, 1937, this patient was seen for discharge and pain in the right ear. The sickness started two weeks previously. The right

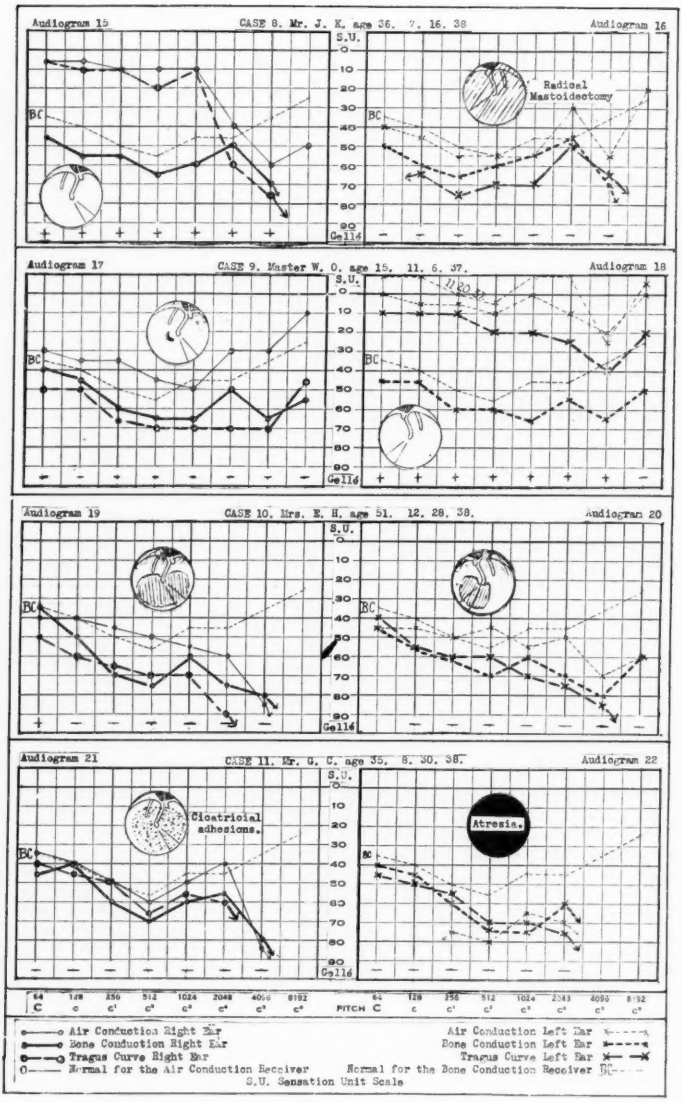

Fig. 3. Audiograms of Gellé negative ears with visible lesions.

ear used to discharge continuously in early childhood, but healed after a simple mastoidectomy was performed by me in 1930. The present inflammation was the first exacerbation after the operation. Otoscopic examination revealed that the right drum-head was inflamed and retracted toward the promontory. There was a central perforation from which oozed a purulent secretion. The left tympanic membrane showed no pathology. Inflammatory congestion of the adenoid tissue, swelling and livid discoloration of both tori were seen through the nasopharyngoscope. The secretion of both nasal fossae was mucous and increased. Moderate inflammatory changes were found also in the pharynx. Four days after the first examination the audiometric tests were performed. By this time the aural discharge disappeared, the fistula diminished and the right tympanic membrane was only moderately discolored. The eustachian tubes were stenotic.

CASE 10.—E. H., female, age 51. Audiograms 19 and 20. The right ear of this patient was continuously discharging since the age of 5 up to the age of 20. Since then the discharge appeared about once every year after a cold. During the last few years also the left ear started to discharge. The present illness lasted about five months. Both ears failed to heal after a cold in August, 1938. On December 28, 1938, the patient complained that during the last two weeks she suffered with attacks of vertigo and had increased noises in both ears. The right ear had a large perforation in the anterior and lower segment of the tense membrane, with many adhesions between the processes of the malleus and the promontory. The left ear had a similar, but smaller, defect in the drum-head. The discharge from both ears was mucopurulent. Nose examination was negative. Mucosa of the nasopharynx was thickened and dry. There were adhesions in the left Rosenmüller fossa. The eustachian tubes were patent.

CASE 11.—G. C., male, age 35. Audiograms 21 and 22. Both ears of this patient were periodically discharging since childhood. The right tympanic membrane was replaced with fibrous bands adherent to the promontory. There was a congenital atresia of the left meatus. Left mastoidectomy was performed when the patient was five years old, and large fistula remained behind the ear. The Gellé test recorded in the audiogram 22 was taken with the Barany tube inserted into the fistula. At the time of the audiometric examination both ears were dry.

The Gellé negative ears of this group can be divided into three types: Type 1 is ear 16 where the middle ear was exenterated and

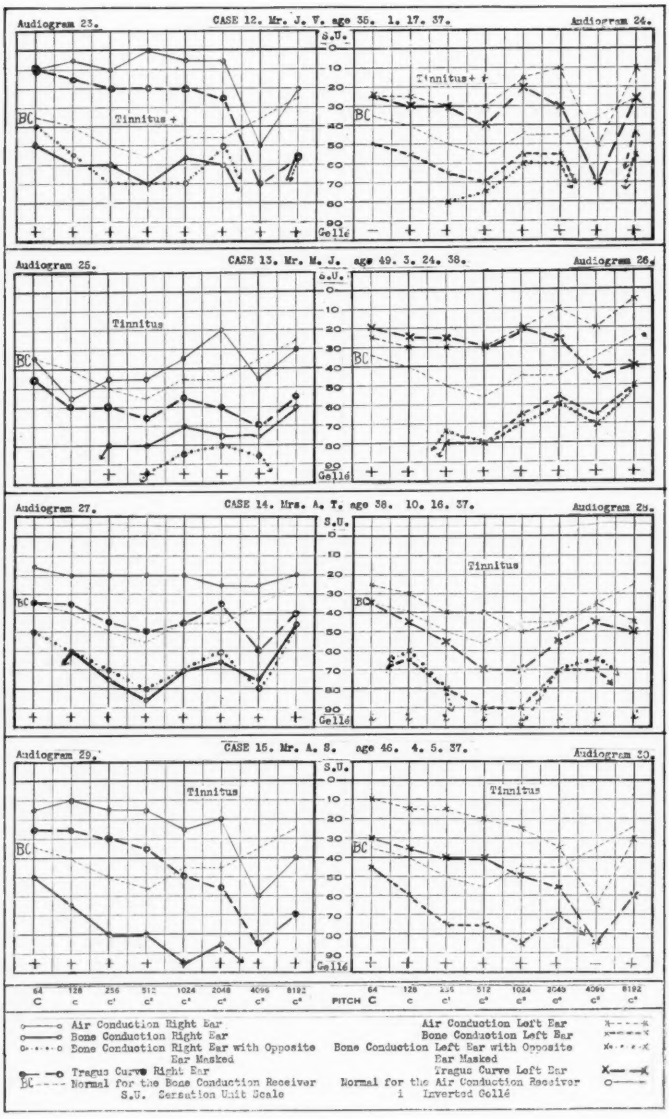

Fig. 4. Audiograms of Gellé positive ears with lesions in the perceptive apparatus.

its mucosa was replaced by skin. Its tragus curve is below the bone conduction curve. Type 2 are ears 17, 19 and 20. They have acute exacerbations of long lasting chronic purulent processes. Their tragus curves are slightly below or on the level of bone conduction curves. Type 3 are ears 21 and 22. They have similar lesions as those of type 2 but were tested in a noninflammatory stage. Their tragus curves are above their bone conduction curves. In all three types the tragus curves are depressed as compared with the Gellé positive normal ears of Fig. 2. The degree of the depression coincides with the amount of disturbance of the mucosa in the middle ear. It is largest where the mucosa is absent in ear 16. Ears with inflamed mucosa have larger impairment of the tragus hearing than ears without the acute inflammatory changes.

Comparison of the air-conduction curves with their corresponding tragus curves, especially in ears 16 and 22, suggests a different character of the two tests. In the atretic ear 22 we may explain the largest depression of the air-conduction curve by the fact that the column of air between the receiver and the middle ear was interrupted by a cutaneous barrière. In ear 16 the air column was not interrupted and the vibrations impinged freely against the promontory. That elevated the air-conduction curve. On the other hand, the continuity of the air column did not help the tragus curve of ear 16 from being depressed below the bone-conduction curve and the interruption of the air path in ear 22 did not prevent the tragus curve from being elevated above the bone conduction. Bearing in mind that both ears are Gellé negative and affected with such changes that the function of the ossicular chain is absent, the unavoidable conclusion is that the meatal air column is of a secondary importance for the level of the tragus curve. That leads to a further deduction of existence of a special main path through which the sounds in the tragus test propagate. The coincidence of the degree of impairment of the tragus hearing with the degree of impairment of the middle ear mucosa suggests that one portion of this path is the mucosa of the middle ear.

THE TRAGUS CURVE IN GELLÉ POSITIVE EARS WITH LESION OF THE PERCEPTIVE APPARATUS

Audiograms of Fig. 3, representing ears with well-defined obstructive lesions, are contrasted by Fig. 4 with audiograms of Gellé positive ears with symptoms pointing to lesions of the perceptive apparatus, such as vertigo, tinnitus, diminished bone conduction and impaired hearing of patient's own voice.

The first and the second patients (cases 12 and 13) had symptoms similar to those described by Shambaugh,[3, 4] namely: decrease of bone conduction for low tones, tinnitus and vertigo. Shambaugh attributes quite an importance to a fourth symptom, diplacousis dysharmonica, and interprets the whole clinical syndrome as perilabyrinthitis. Unfortunately, Dr. Shambaugh's articles did not come to my attention at the time when these patients were examined and the diplacousis test was not performed. Other authors acknowledge only three symptoms: Loss of bone conduction, tinnitus and vertigo, and classify the trias as Ménière's disease. The third patient (case 14), as well as the first two, would then be Ménière cases, though there are some substantial differences between them. Wright[5, 6] concludes in a study of 73 cases that such symptoms are due to focal labyrinthitis similar to focal iritis. Crowe[7, 8] believes that the irritation is due to circulatory or chemical disturbance in the endolymph and not to foci of infection in the ear or in other parts of the body. However, his and his collaborators' studies failed to localize definitely the site of the lesion, and in some patients the tinnitus persisted even after section of the acoustic nerve. Other authors diagnose cases with similar symptoms as toxic neuritis, catarrhal otitis media with involvement of perilabyrinth or as circulatory disturbances in the cochlea. Consensus of opinion is that it is a disease of some part of the perception system of the ear.

The third patient of Fig. 4 (case 14) differs from the first two patients by his Gellé reaction, which is inverted, and also by a maximum depression of the bone conduction not in low, but in middle frequencies. It is interesting that this patient's vertigo could be provoked by consumption of alcohol.

The fourth patient (case 15) is different type again: The patient suffers with tinnitus but has no history of vertigo. His loss of bone conduction increases with the pitch. Besides that he exhibits a marked defect in 4096 d.v. similar to that of case 12. His pathology is probably that described by Crowe,[9, 10] namely, partial atrophy of the cochlear nerve in the osseous spiral lamina of the first turn of the cochlea (decrease of hearing with increasing pitch) combined with a total atrophy of the organ of Corti in some part of the basal turn (the defect in 4096 d.v.).

Case 12.—J. V., male, age 35. Audiograms 23 and 24. On November 12, 1937, this patient reported that two weeks previously he suddenly noticed buzzing in both ears. It lasted for five hours. Four days before the visit the buzzing started again and became

increasingly annoying. On November 11, 1937, he had a short, disagreeable attack of vertigo. Previous aural history was negative except that the patient had "pain and abscess" in the left ear at the age of 13. Past medical history revealed no acute infections in adult age and no trauma. Patient was a heavy smoker. There was no abuse of alcohol, quinine or of coal-tar derivatives. Examination on November 12, 1937, revealed scarlet red discoloration of the septum, of nasopharynx and of tori. Nasopharynx also showed swelling and increased secretion. Tympanic membranes had normal appearance. Eustachian tubes were moderately stenotic. There was no spontaneous nystagmus. The patient was advised to limit smoking and nasopharyngeal therapy was introduced. After five days the symptoms somewhat changed. The vertigo appeared again several times but was less severe. The noise was more on the left side and resembled a hum of a shell applied to the ear. There was less swelling in the nasopharynx. Eustachian tubes were patent. Audiograms 23 and 24 were taken in this stage. One week later the patient reported complete disappearance of tinnitus and vertigo. His audiograms showed a complete recovery of masked bone conduction.

CASE 13.—M. I., male, age 49. Audiograms 25 and 26. Mr. I. was treated by me in July, 1932, for noise in the right ear and for attacks of vertigo. My record shows that the patient had a nasopharyngeal catarrh and swelling of the right eustachian orifice. The subjective and objective symptoms cleared up in two weeks. On March 24, 1938, the patient visited me again. He had no difficulties for six years. Three weeks ago a noise "like a buzz and a whistle" started in the right ear. Attacks of dizziness appeared several times a day. Past medical history was essentially negative. There was no abuse of alcohol and tobacco. Examination revealed impacted tympanic membrane on the right side, turgescent middle and inferior turbinates on both sides and enlarged tonsils with inspissated secretion in the crypts. The nasopharyngeal mucosa was of purplish hue, thickened and covered with flocculent mucopurulent secretion. Inflation of right eustachian tube was difficult but finally the air passed through with high-pitched tone. The left tube showed no stenosis. There was no spontaneous nystagmus. The blood pressure was 145/80. Urine, Wassermann and teeth x-ray were negative. Audiograms 25 and 26 were taken on March 24, 1938. On April 1st an improvement of symptoms was reported and the air conduction in both ears was better. Two weeks later a relapse followed. The noise and the dizziness failed to clear during

the following two months. The eustachian tubes, however, were patent. The tonsillar and the nasopharyngeal infection resisted therapy. The patient reported later that the tinnitus and vertigo subsided during the hot summer months.

CASE 14.—A. T., female, age 38. Audiograms 27 and 28. Mrs. T. complained on October 16, 1937, of severe noises in the left ear for the past two months. During the same period she suffered with attacks of vertigo which appeared sometimes spontaneously and always after an alcoholic drink. Similar attacks of vertigo and tinnitus were experienced several times during the past few years. The left ear used to ache occasionally but there was no history of aural discharge. There was no deafness in the family. Both drum-heads had normal appearance. The nose was not obstructed. The nasopharynx was deeply vaulted and congested. The eustachian orifices had normal appearance. The right salpinx was patent, the left was stenotic. There was no spontaneous nystagmus. Audiometric examiniation was performed on October 16, 1937. Ten days later, after nasopharyngeal therapy with mild silver-protein, a record of improved hearing in the left ear was obtained. Two weeks later the patient reported improvement of subjective symptoms.

CASE 15.—A. S., male, age 46. Audiograms 29 and 30. The patient noticed buzzing in both ears for several years. Lately he observed hardness of hearing and stabbing pains in the left ear. There was no history of aural discharge, dizziness, repeated colds, smoking, or gastrointestinal disorders. On April 5, 1937, the left tympanic membrane was moderately congested, the right side was normal. Left nasal fossa was obstructed by deflected septum and by hypertrophic inferior turbinate. The nasopharyngeal mucosa was thickened, the tori appeared infiltrated. The tonsils were enlarged. X-ray of the teeth showed apical abscesses on the right upper lateral incisor and on the right upper first molar. Extraction was suggested but the patient refused to follow the advice. Inflation of the tubes was not performed. Within three weeks the congestion of the left ear disappeared but there was no improvement of the head noises.

The tragus curves of the first two cases (12 and 13) show an interesting course: They approach (ear 23, 24, 25) or cross (ear 26) the air conduction curves in the low tones. At the same time the ears with greater impairment of hearing (24, 25, 26) show loss

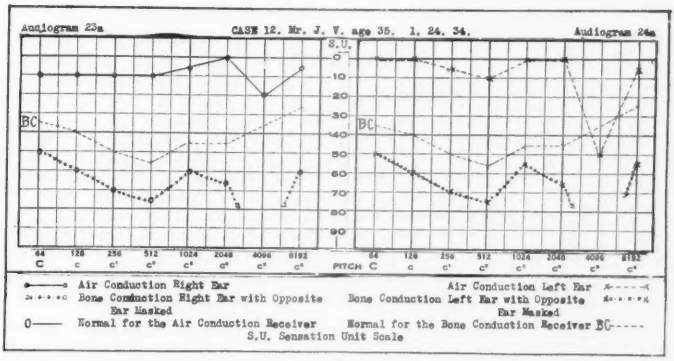

Fig. 5. Improvement of hearing in Case 12 of Fig. 4 after disappearance of tinnitus and vertigo.

of bone conduction in low ranges and divergence of the bone-conduction curves from the tragus curves.

In case 12 the loss of low tones by bone conduction was on the left side only and was detected by masking of the opposite ear. At the time of the audiometric examination the patient localized the tinnitus into the left ear. The island of deafness around 4000 d.v. in this case had evidently no relation to the acute clinical symptoms, because it remained after the disappearance of the tinnitus and vertigo (audiograms 23a and 24b, Fig. 5). The patient had originally noises in both ears and stenosis of both eustachian tubes. The only audiographic sign of affection on the right side is the approach of the tragus curve to the air-conduction curve in the low tones.

Case 13 actually represents a more advanced stage of the same disease. Apart from the deafness island the ear 26 exhibits losses of hearing almost equal to those of ear 24. But ear 26 is the better ear of patient 13, who had tinnitus only in the right ear. Both tragus curves of this patient have a diagonal position between the bone-conduction and air-conduction curves, i. e., they show greater losses in the high tones than in the low tones. The tragus curve in ear 26 is depressed in high tones only and normal for the low tones. In more advanced stage of ear 25 the tragus hearing becomes depressed in the whole extent, but more so in the high tones.

Pathologic interpretation of these audiograms will not be attempted in this essay. Suffice here to recognize the interesting fact that in spite of a great loss of low tones by bone conduction the tragus curve remained unimpaired in certain moderately advanced cases of Ménière's disease, and, when the tragus conduction became affected, the maximum decrease appeared not in the region of the acoustic spectrum where the bone conduction was deficient, but on the opposite end.

Case 14 does not differ clinically from the previous two cases. The tinnitus was unilateral, the vertigo appeared periodically, there was some nasopharyngeal and tubar pathology like in cases 12 and 13. The audiograms showed that the affection was also bilateral. The active side, however, shows three areas of bone-conduction loss: the highest tone, the lowest tone and two tones of the speech frequency 512 d.v. and 1024 d.v. (Audiogram 28). The tragus and the air-conduction curves do not show so distinctly their typical low-range approach. They run parallel to the bone-conduction curve. The most striking feature is the inverted Gellé—the patient heard better if the pressure in the external meatus was increased or decreased. The inactive ear (audiogram 27) shows a linear depression of air-conduction and bone-conduction curves and an increased loss of high tones in the tragus curve. Case 14 may represent a separate pathologic entity or only a different manifestation of the same disease which affected the two previous patients. The importance of the tragus curve lies in the fact that its position is one of the differential characteristics in a group of clinically similar ears.

Audiograms 29 and 30 of case 15 are similar to the audiograms 10 and 11 of Fig. 2. All four show a gradual decrease of all three curves with increasing pitch. Their bone conductions are approximately on the same level. The difference is that the patient No. 15 is thirteen years younger than patient No. 5, and twenty years younger than 67-year-old patient No. 6. Furthermore, patient No. 15 had a long lasting tinnitus in both ears, while the older ears, Nos. 10 and 11, had no history of noises. The reader will notice that the right ear of the patient No. 5 and the left ear (No. 12) of patient No. 6 exhibit audiographic features similar to cases 12 and 13 of Fig. 4. They are both tinnitus ears. Their masked bone conduction was not taken, but 64 d.v. is lost from the bone conduction. Though there is no history of vertigo, the audiograms are very suggestive of the same pathology as that of cases 12 and 13. Their sickness is, however, superimposed on senile changes.

In spite of similar audiograms the pathology of ear 29 (Fig. 4) could hardly be explained as premature senility. The tinnitus and the apical dental abscesses furnish a better interpretation. It might have been a toxic process of focal origin, affecting the spiral ganglion. Ear 30 of the same patient has the same basic defect on which a mild inflammatory process in the middle ear is superimposed. That explains the increased bone conduction and the depression of the other two curves.

Study of the Fig. 4 demonstrates that the tragus conduction is independent from the bone conduction and that the tragus test does not eliminate the necessity of bone-conduction masking. The loss of low tones in ear 24 could not be discovered without elimination of the opposite ear. The Rinné test in ear 24 would have been entirely misleading, because its bone-conduction part would have been actually a test of the opposite ear 23. The masking in these tests was performed by noise produced by an electric buzzer and by a current of air conducted to the ear from a specially constructed instrument described previously by the author.[11]

THE TRAGUS CURVE IN OTOSCLEROSIS AND IN CHRONIC CATARRHAL DEAFNESS

Figure 6 represents audiograms of four carefully selected patients in which a clinical differentiation could be made between otosclerosis and chronic catarrhal deafness. With an exception of ear 38, all ears were totally or partially (ear 32 and 35) Gellé negative.

The first two patients (cases 16 and 17) were affected with otosclerosis. Case 16 was incipient, case 17 was advanced. Both patients had positive family histories of deafness, negative nasal and nasopharyngeal findings and patent eustachian tubes. Ears 31, 33 and 34 were affected with tinnitus and had pink, translucent membrana tensa. Both patients were under observation for a long time. They had no attacks of vertigo and no frequent recurrences of nasal infections.

The last two patients (cases 18 and 19) had chronic catarrhal deafness. Both patients had negative family histories of deafness, positive nasal, nasopharyngeal, and otoscopic findings and stenotic eustachian tubes. Ears 36, 37 and 38 were affected with tinnitus. Both cases had a history of frequent head colds and infections of their upper respiratory passages were seen during the course of observation.

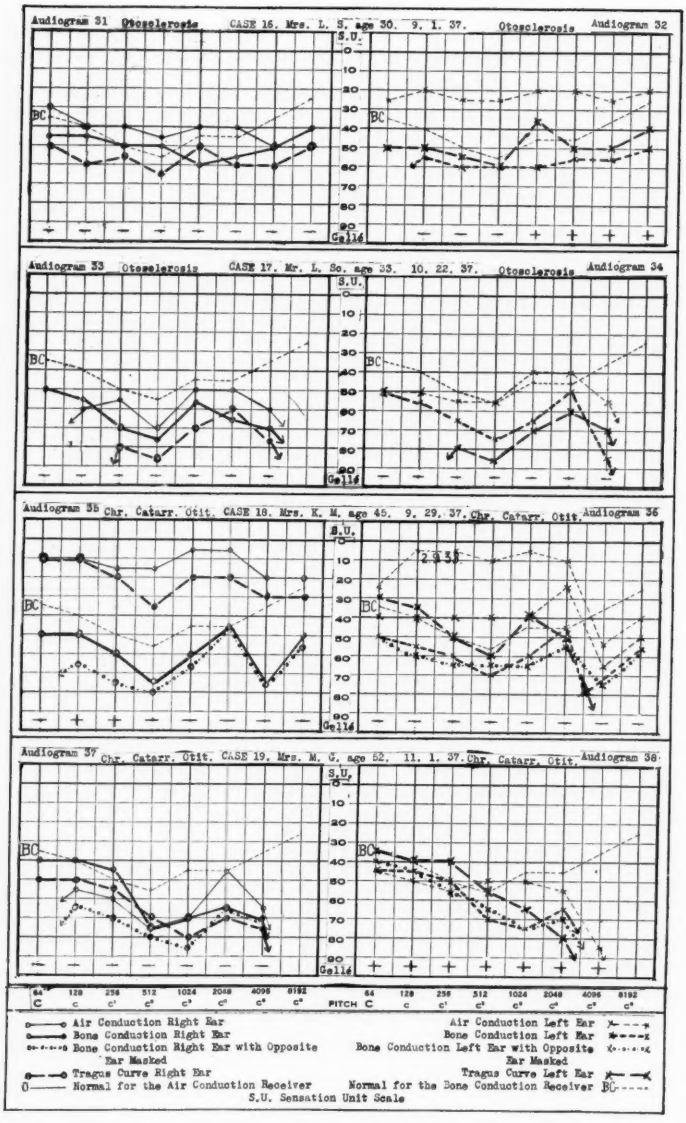

Fig. 6. Audiograms of two cases of otosclerosis and of two cases of chronic catarrhal deafness.

CASE 16.—L. S., female, age 30. Audiograms 31 and 32. Mrs. S. noticed loss of hearing in the right ear three years before she visited me on September 23, 1936. Noise in the right ear started about one year after the impairment of hearing. There was no history of vertigo, pain or discharge from the ears. Both grandparents were hard of hearing. Right membrana tensa showed pink translucency. The left ear was otoscopically negative. Nasal passages, nasopharynx and eustachian orifices appeared normal. The patient was under observation for 18 months. The acuity of hearing in the left ear varied during the period of observation within about 10 to 20 sensation units by air conduction. The right ear showed very small variations.

CASE 17.—L. Sc., male, age 33. Audiograms 33 and 34. This patient was hard of hearing for 15 years. Tinnitus started on the right side at the age of 20, on the left side a few years later. There was no history of aural discharge or of acute inflammations. One sister and one aunt were hard of hearing. Examination of ears, nose, nasopharynx and pharynx revealed no pathology except pink and translucent drum-heads in both ears. There was no obstruction of the eustachian tubes. Mr. Sc. was under observation for a period of six months. The air-conduction curves of both ears showed only small variations.

CASE 18.—K. M., female, age 45. Audiograms 35 and 36. On September 25, 1937, Mrs. M. gave a history of tinnitus in the left ear lasting two years and of blowing noise in the right ear of recent origin. She suffered with frequent head colds associated with occasional stabbing pains in both ears. There was no history of aural discharge or of vertigo. No other member of the family was affected with deafness. Both drumheads were retracted and showed a distorted light-reflex. The nasal secretion was serous and increased. The nasopharynx was thickened and covered with mucopurulent, flocculent secretion. The right eustachian tube was patent, left stenotic. The patient was under observation and treatment for six months. The improvement of hearing in the left ear after five months is shown in the audiogram 36 by a curve dated February 9, 1938. At that time the tinnitus on the right side was absent and on the left side appeared only occasionally. Both ears became Gellé positive.

CASE 19.—M. G., female, age 52. Audiograms 37 and 38. This patient was hard of hearing for 12 years, but on November 1, 1937, she complained that about one month before, her hearing

became much worse. Both ears were affected with noises of varying intensity for several years. Lately the tinnitus in the right ear increased. There was no history of vertigo, aural pain or discharge. Family history of deafness was negative. Both ear-drums showed calcified areas, retraction of flaccid and tense membranes and loss of luster. Nasal mucosa was hypertrophic, septum thickened and breathing difficult. Mucosa of the pharynx and nasopharynx was atrophic and covered with inspissated secretion. Both eustachian tubes were stenotic. Inflation on the right side succeeded only after cocainization. The patient was under observation and treatment for one month. The tinnitus in the right ear improved. The right air-conduction curve increased 15 to 20 sensation units, the left 5 to 10 sensation units. The right ear remained Gellé negative.

The first four audiograms of Fig 6 (audiograms 31 to 34) demonstrate the influence of osseous occlusion of the oval window on the tragus curve. In ear 32 the occlusion was not complete—the Gellé was positive in high tones; yet the tragus curve is depressed almost to the level of bone conduction. The air-conduction curve suggests much less obstruction. In ear 31 we may assume a complete osseous ankylosis of the stapes. The tragus curve is below the level of bone conduction. The otosclerotic process in patient 16 did not affect the perceptive elements, because the bone conduction is good. In ears 33 and 34 the osseous ankylosis of the stapes is complete like in ear 31, but the perceptive elements are damaged, as witnessed by decreased bone conduction and loss of high tones. The tones conducted from the tragus did not reach the apical turn of the cochlea, as manifested by the loss of low tones on both tragus curves.

Audiograms 35, 36, 37, 38 are records of ears affected with chronic catarrhal otitis media. The total (ears 36 and 37) and partial (ear 35) ankylosis of the stapes is presumably not of osseous but of fibrous character. The partial ankylosis of ear 35 did not affect the tragus conduction. The position of the tragus curve in this ear exhibits a noticeable difference from that of the otosclerotic and partially Gellé negative ear 32. Even in the totally Gellé negative ears 34 and 37 the tragus curves are above the bone-conduction curves, while in the totally Gellé negative otosclerotic ears 31, 33 and 34, the tragus curves are below bone-conduction curves.

The conclusion of this comparison is that an osseous block in the oval window offers a much greater obstacle to the propagation

of the sound from the tragus than a fibrous ankylosis of the footplate.

The relations of the air-conduction curves to their respective bone-conduction curves show no difference between the osseous and fibrous ankyloses. All the air-conduction curves are essentially above their bone-conduction curves. Only in the two lowest tones of the advanced otosclerotic ear 33 the air-conduction curve crosses below the bone-conduction. Such crossing is also seen in the catarrhal ear 38, which is, however, Gellé positive. Evidently the depression of the air-conduction curves below the bone-conduction thresholds of the low tones is not in a primary relation with the mobility of the stapes-plates in these two ears.

Observation of the relations between the tragus and air-conduction curves is more interesting. In all four otosclerotic ears the air-conduction curves remained above the tragus curves. But in the low tones of all four catarrhal ears the air-conduction curves approached or crossed below the tragus curves.

We have seen a similar crossing or approach of these two curves in cases of complete Ménière's syndrome (cases 12 and 13) of Fig. 4, and also in several ears of Fig. 2 (audiograms 3, 6, 7, 9, 12) and of Fig. 3 (audiograms 15, 18, 20, 21, 22). These ears furnish a great variety of middle ear, cochlear and other pathologies which should be analyzed in order to find a common denominator responsible for the relation of the two curves.

THE INTERPRETATION OF THE TRAGUS-AIR-CURVE CROSSING

Table I represents a survey of 20 ears belonging to 14 patients whose histories and audiograms have been studied in this essay. All of them show the crossing of air-conduction curves below the tragus curves in the low-tone range. Assuming that this "tragus-air-curve crossing" is a symptom of some specific pathology in some part of the ear, the negative or normal findings listed in the summary of the table will help us to eliminate several regions of the hearing organ from the correlation. Thus the pathologic changes in the tympanic membrane are certainly not responsible for the crossing of the curves, because it occurred in audiograms of eleven ears with normal drumheads.

Similarly, the stapes and the eustachian tubes are beyond suspicion, because the crossing occurred in fourteen ears with mobile stapes and in nine ears with patent eustachian tubes. Tinnitus and

Case No.	Ear No.	Tympanic Membrane	Middle Ear	Stapes	Eustachian Tube	Bone Conduc. Low Tones	Bone Conduc. High Tones	Tinnitus	Vertigo	Present or past evidence of nasal, nasopharyngeal or aural pathology	Nasophar.	Remarks
2	3R	Normal	?	Mobile	Stenotic	Dimin.	Normal	–	–	History of right purulent antritis	–	Bell's palsy R.
3	6L	Normal	?	Mobile	Stenotic	Dimin.	Dimin.	+	–	Congested naso-phar. Impacted middle turb.	+	
4	7R	Perforated	Granuloma	Mobile	Patent	Dimin.	Dimin.	–	+	Granuloma and cholesteatoma.	–	
5	9R	Normal	?	Mobile	Patent	Dimin.	Lost	+	–	Hypertr. nasophar. Frequent colds.	+	
6	12L	Normal	?	Mobile	Patent	Dimin.	Lost	+	–	Hypertr. nasopharyngitis.	+	Diabetes
8	15R	Normal	?	Mobile	Patent	Normal	Dimin.	–	–	Radic. mastoidectomy on the opposite side.	–	Working in noise
9	18L	Normal	?	Mobile	Stenotic	Normal	Normal	–	–	Acute nasophar. Recurr. pur. of med. dextra.	+	
10	20L	Perforated	Discharge	Immobile	Patent	Normal	Dimin.	+	+	Chronic nasophar. Frequent colds.	+	
11	21R	Perforated	Adhesions	Immobile	Not exam	Increa	Dimin.	–	–	R: Chr. pur. ot. med.	+	
	22L	Atresia	Discharge	Immobile	Not exam	Increa	Dimin.	–		L: Ch. pur. ot. med. atresia of meatus.		
12	23R	Normal	?	Mobile	Stenotic	Normal	4000Def.	+	+	Sub-acute nasopharyngitis.	+	
	24L	Normal	?	Mobile	Stenotic	Lost	4000Def.	+				
13	25R	Retracted	?	Mobile	Stenotic	Lost	Dimin.	+	+	Hypertrophic nasophar. Chr. tonsillitis.	+	
	26L	Normal	?	Mobile	Patent	Lost	Normal	–				
18	35R	Retracted	?	Part-mob.	Patent	Dimin.	4000Def.	+	–	Chronic nasophar. Frequent colds. Sub-acute rhinitis.	+	
	36L	Retracted	?	Immobile	Stenotic	Normal	4000Def.	+				
19	37R	Retracted	?	Immobile	Stenotic	Dimin.	Dimin.	+	–	Atrophic nasopharyngitis.	+	
	38L	Retracted	?	Mobile	Stenotic	Normal	Dimin.	+				
20	39R	Normal	?	Mobile	Patent	Normal	Dimin.	+	+	Chr. hypertr. rhin. and nasophar. Frequent colds.	+	
	40L	Normal	?	Mobile	Patent	Dimin.	Dimin.	+				
Total	Total	Normal 11 Perf. 3 Retr. 5 Atres. 1		Mobile 14 Immob. 5 Part-mobile 1	Patent 9 Steno. 9 Not exam. 2	Norm. 7 Dim. 8 Lost 3 Incr 2	Norm. 3 Dim. 11 Lost 2 4000 4	13 7	5 9	+ –	11 3	
14	20	Total 20		Total 20	Total 20	Tot 20	Tot 20	20	14	Total	14	

Table I. Analysis of 20 ears showing a crossing of the air-conduction curve below the tragus curve.

vertigo show no better correlation. Seven ears with the tragus-air-curve crossing had no noises and nine patients had no vertigo.

The bone conduction for the low tones should be scrutinized very carefully. It is well known that the air-conduction receiver has a bone-conduction error, that is, the tone reaches the homolateral cochlea not only through the conductive apparatus but also through the skull. If the bone conduction for the low tones is diminished or lost, also the bone-conduction component of the air-conduction curve is diminished or absent. And if there should be less bone-conduction error in the tragus test than in the air-conduction test the tragus and air-conduction thresholds might cross. However, the crossing occurred also in two ears (21 and 22) with increased bone conduction for low tones and two additional ears of such type are seen in Fig. 8. Besides that, the approach of the two curves is found also in two ears (3 and 23), where the air conduction for low tones is very near the normal threshold. In such intensities the cranial component of the air-conduction test is nil. This has been established by a simple experiment which is described in the following paragraph. We have to conclude that the bone conduction for the low tones is not a factor in the tragus-air crossing.

Evaluation of the cranial component of the air-conduction threshold in ears with normal hearing: The air-conduction receiver is applied over the ear with the audiometer switched off. Over the mastoid process of the same side a vibrating tuning fork is held until it is no more heard. The frequency of the tuning fork is now switched into the receiver with intensity about 10 decibels above the observer's threshold. The still vibrating fork is now alternatingly applied and removed from the mastoid process. A slight increase in the loudness is observed whenever the fork is in contact with the bone. Next the observer should find his threshold when the receiver is held close to, but not in contact with the ear. The above experiment is then repeated while the ear again listens to a tone 10 decibels above the second threshold. No increase in loudness is now caused by the sub-threshold vibrations of the tuning fork. Evidently the increase occurred only when the ear was closed by the receiver. Consequently, if any sub-threshold bone-propagated energy enhances the hearing through the air-conduction receiver, it so happens by action of the cranio-meato-tympanic, and not by the cranial, component of the bone conduction. Such interference, if any, still represents the meatal function and cannot be responsible for the low tone depression of the nearly

normal air-conduction hearing in ears with loss of low tones by bone conduction.

Bone conduction for the high tones seems to have a good alibi in our investigation, since it occurs on the opposite end of the acoustic spectrum. Yet there are only three ears which are classified as normal in this range against 17 which exhibit some sort of defect. All three had very little evidence or history of pathology. Ear No. 3 appeared healthy in all respects except for a stenosis of the eustachian tube. It belonged to a patient with a homolateral Bell's palsy. Ear No. 18 was the good ear of a patient recovering from an attack of recurrent purulent otitis media in the opposite ear. It had salpingeal stenosis but no other evidence or history of pathology. Ear No. 26 was the better ear in a Ménière's case. Its audiogram shows loss of low tones by bone conduction, and the patient had nasopharyngeal and tonsillar infection. But its eustachian tube was patent and it showed no physical evidence of pathology.

The conclusion about the relation of the loss of high tones by bone conduction to the crossing of the tragus and air-conduction curves in the low tones should be guarded. The series is too small to have a statistical value. But there is a clinical evidence that the high tones remained intact only in ears with minimum pathology. We may suspect that the same process which caused the crossing sign may in later stages be responsible for disturbances of high tone hearing. Such relation, however, is not of acoustic but of clinical character. Considering now both, the low and the high tones of bone conduction, we may be satisfied that the tragus-air crossing is not caused by the pathology in the perception organs of the ear.

If the crossing is of pathologic origin at all—and such seems to be the case, considering the patients of Fig. 2, where the pathology was unilateral with the opposite ear normal—it must be pathology of the conduction. The external ear and the tympanic membrane having been excluded, the site of the suspected lesion is limited to the tympanic cavity.

We have found that the meatal air column is of secondary importance for the level of the tragus curve as related to the bone conduction, and that part of the tragus hearing path is probably the mucosa of the middle ear. The evidence for the "membranous" character of the tragus path was further corroborated by the observation that the level of the tragus curve in relation to the bone-conduction curve was better when the ankylosis of the stapes

was of fibrous character and worse if the obstruction in the oval window was osseous. The levels of the air-conduction curves as related to the bone-conductions did not follow the levels of the tragus curves, but remained above the bone-conduction curves, with exception of one atretic ear, No. 22. That indicates that the sound travels a different path in tragus test than in the air-conduction test.

In the air-conduction test the main portion of the acoustic vibrations is admittedly intercepted by the tympanic membrane and conducted over the ossicular chain to the oval window. A secondary transmission to the cochlea occurs from the tympanic air space through the promontory and the round window.

So far we have found no evidence of the amount of acoustic energy transmitted over the ossicular chain and through the tympanic air space in the tragus test. All that we know is that part of the acoustic energy is transmitted from the tragus to the membranous structures of the middle ear. The tympanic air, being a part of the air column, is probably of secondary importance even in the tragus transmission (see audiogram 16). Now if the air-conduction curves of certain ears cross below their tragus curves, it may be a result either of impairment of the ossicular transmission in the air-conduction test or of enhancement of the membranous transmission in the tragus test or of both. Comparing the ears in cases with unilateral cross-sign (Fig. 2), namely, in case 2, ear 3, with ear 4; in case 3, ear 5, with ear 6, and in case 5, ear 9, with ear 10, we see that the air-conduction curves of the diseased ears show a decrease of air-conduction curves and no increase (ears 3 and 6) or a slight decrease (ear 9) of the tragus curves. Also in Fig. 4, comparison of the worse ears with their better ears (ear 24 with ear 23, and ear 25 with ear 26), shows that the tragus curves of the worse ears are not elevated, but more or less depressed—the depression of the air-conduction curves, however, being always greater than that of the tragus curves. The evidence points clearly to the insufficiency of the ossicular chain as the factor responsible for the tragus-air crossing. Furthermore, because the crossing occurs in Gellé negative as well as in Gellé positive ears, the stapes-plate can be exonerated and the conclusion can be more specific:

The crossing of the air-conduction curve below the tragus curve in the low tones is caused by limitation of movements in the ossicular articulations.

Such conclusion should be supported by evidence other than acoustic phenomena. In the group of ears listed in Table I, the majority of middle ears were inaccessible to direct examination and are marked with a question mark in the middle ear column. In those cases the evidence should be at least circumstantial: All patients of the series had some past or present nasal, nasopharyngeal or aural pathology. It is interesting that all cases except three had nasopharyngeal involvement. Spreading of infection from the nasopharynx into the delicate and loose tissues of the epitympanum where the ossicles articulate is a well admissible clinical possibility. The three cases which showed no nasopharyngeal infection also had sufficient clinical indications of possible ossicular ankyloses. Ear 7 of case 4 had a cholesteatoma in the epitympanum. Patient No. 8 had a history of chronic purulent otitis media in the opposite ear. Such process is never strictly unilateral. Besides that, he was working in the noise of a pneumatic hammer. That was certainly quite a strain on the ossicular joints, especially if they were previously slightly damaged. Case No. 2 is very interesting. With no aural history and no subjective ear symptoms the patient exhibits three homolateral symptoms: Bell's palsy, loss of low tones by bone conduction similar to that of Ménière cases 12 and 13, and the crossing sign interpreted here as ankylosis in the ossicular chain. Definite history of homolateral purulent antritis added to the above trias offers sufficient ground for speculation about the pathogenesis and the site of lesion in the Bell's palsy and in the Ménière's syndrome.

Assuming that the tragus-air crossing is a symptom of ossicular ankylosis, we should yet inquire whether the damaged articulation has any influence on the hearing threshold of low tones in the tragus test, i. e., whether any substantial portion of the low frequency vibrations propagates in threshold intensities from the tragus over the ossicular chain. We have seen several depressed tragus curves in Gellé positive ears (6, 7, 9, 24, 25, 28, 34, 38). In all of them with the exception of ears 30 and 38, also the bone conduction was depressed so that no conclusion can be drawn whether the decrease of tragus hearing was caused by ankylosis or by cochlear involvement. Ear 30 had an acute inflammation in the tympanum which might have changed the conductive qualities of the mucosa. Ear 38 is rather exceptional, showing fairly good bone conduction, especially in low tones, deep defect in air conduction and mobile stapes. Interpretation of its audiogram may be that of complete ankylosis in the ossicular articulation with freedom of the stapedial plate. In that event, the other ankyloses should be classified as partial limi-

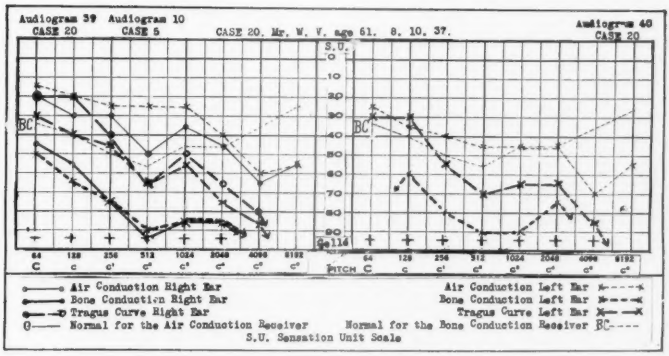

Fig. 7. Chronic catarrhal deafness in the right ear of Case 20 compared with normal hearing in the left ear of Case 5. Both patients belong to the same age-group.

tations of movements in the ossicular joints, or partial ankyloses. But there is probably one more factor which modifies audiogram 38, namely, the bone conduction is enhanced by a block in the round window. Then the tragus-bone curve-distance in this ear is not a specific expression of ossicular ankyloses.

Another insight into the question of tympanic tragus-path is furnished by Fig. 7. It represents audiograms of case 20, the right ear of which is compared with the left ear of case 5.

CASE 20.—W. V., male, age 61. Audiograms 39 and 40. This patient has been under my observation since 1934. At that time he gave a history of difficult nasal breathing for many years, ringing in both ears for the past three years and periodic attacks of vertigo associated with occipital headaches for the past two years. On May 7, 1934, the otoscopic examination was negative. Both nasal fossae were obstructed by thickened septum and by hypertrophic turbinates. Tonsils were removed several years ago. Submucous resection of septum was performed in May, 1935. After the operation and persistent therapy the nasal breathing improved and the attacks of vertigo occurred only very rarely. The patient, however, remained predisposed to head colds. The mucosae of the nose, and especially of the nasopharynx, continued to be very much hypertrophic. Translumination of the sinuses showed 2-plus shadow of maxillary cavities. Hearing by air conduction gradually de-

clined in both ears during the course of observation. On August 10, 1937, when the audiograms 39 and 40 were taken, the eustachian orifices appeared normal and the tubes were patent. The tympanic membranes never showed any changes. The head noises persisted, but became less troublesome. Spontaneous nystagmus was never observed. Blood pressure never exceeded the physiologic limits. Urine, Wassermann and dental x-rays were negative.

Patient 20 is similar to patient 5 in the respect of age, sex, unilateral bone-conduction decrease (ears 9 and 40), history of frequent colds, otoscopic and eustachian findings. Audiogram 9 is similar to audiogram 40, audiogram 10 resembles audiogram 39 in every respect except in the low-tone level of the three curves. In the low ranges ear 10 has worse bone conduction, worse tragus conduction but better air conduction that ear 39. Both ears are Gellé positive. Ear 10 was classified as normal, the gradual decrease of hearing with the increase of pitch being interpreted as physiologic loss of hearing in the patient's age of 59. The bone-conduction and tragus curves of ear 39 can also be interpreted as showing only declines physiologic for the age of 61 years. But the air conduction shows too much depression in the entire hearing range. Something interfered with the transmission of sound from the air-conduction receiver. Patient 20, who had the worse air conduction, had much more marked nasal and nasopharyngeal congestion and hypertrophy. He had longer history and more severe general symptoms of headaches, vertigo and tinnitus. We are justified to assume that he had some mucosal and ankylotic changes in the epitympanic space. In spite of that his tragus curve did not decline in the low tones. On the other hand, the tragus curve of patient 5 paralleled the depression of the low tones by bone conduction. These observations indicate, first, that partial ossicular ankylosis did not affect the tragus hearing in the ear 39, and secondly, that in ear 10 the low ranges of the tragus curve were not helped by the good ossicular chain but registered the same decline of cochlear hearing as the bone conduction.

From this analysis we should not infer the tympanic tragus path without bearing in mind that both, the tragus curve and the air-conduction curve, are records of threshold perceptions. The threshold in the tragus test appears to be approached through the membranous tympanic route and in the air-conduction test through the ossicular route. It is a theoretical possibility that the threshold intensity in the tragus test comes to effect in the oval window as a sum of two components, one major, representing the mucosal

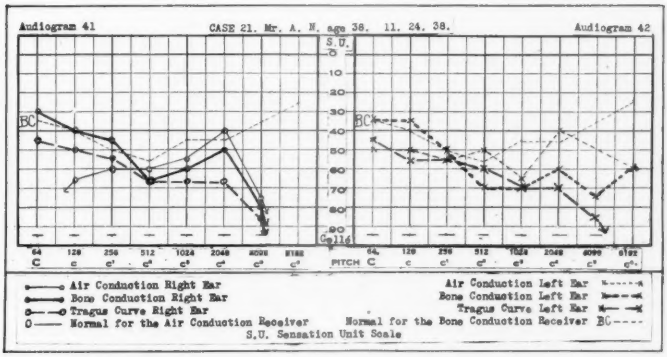

Fig. 8. A case of otosclerosis showing tragus-air-curve crossing in low tones.

vibrations, the other minor, representing the ossicular vibrations. Such a hypothesis does not find support in our analysis of cases 20 and 5. However, when high intensities have to be applied in order to reach the tragus threshold, as in ear 38, there may be some ossicular element in the tragus test. Then the deduction about the propagation of the sound from the tragus through the middle ear is as follows: The low intensities reach the tragus-hearing threshold through the mucosa of the middle ear. High intensities probably propagate also through the ossicular chain.

The presence of the tragus-air-curve crossing in the chronic catarrhal ears of Fig. 6, and its absence in the otosclerotic ears, should not lead to generalization. This sign should not be ascribed an absolute diagnostic value differentiating between the chronic catarrhal deafness and the otosclerosis. While it can be assumed that ankylosis of the ossicles is present in all ears with chronic catarrhal changes, it also is a fact that otosclerosis causes secondary inflammatory reaction in the epitympanum and around the ossicles. A good histologic section of such a case can be found in the chapter on otosclerosis in the Atlas of Otolaryngologic Pathology.[12] An audiogram of an otosclerotic patient with tragus-air-curve crossing is represented by Fig. 8.

CASE 21.—A. N., male, age 38. Audiograms 41 and 42. Hardness of hearing was first observed by this patient at the age of 26. He had persistent noises in both ears. There was no his-

tory of earaches, suppurations or of vertigo. Father, grandfather and one uncle were hard of hearing. Ears, nose, nasopharynx and the eustachian tubes showed no pathology. Audiographic observations on this patient were made by me since 1934. Gradual decline of hearing, especially in the right ear, was observed.

THE TRAGUS TEST AND THE RINNÉ TEST

In a previous study, the author[13] published a modification of the rule of Fowler,[14] who was the first to offer an interpretation of audiograms by the comparison of air-conduction and bone-conduction curves according to a rule analogic to the rule of Rinné. The author's modification is here reproduced:

1. If all changes in both A. C. and B. C. curves occur in the sense of decreased acuity against the normal of the audiogram, the loss of hearing is probably caused by the perception factor. This factor may not always be of pathologic character and may mean only a physiologic variation if the losses are commensurable with the average acuity for the patient's age, ear training, intelligence, mentality, etc.

2. If the bone-conduction curve presents in one or more frequencies a small decrease, normal, or increase above the normal, while the air-conduction curve manifests in the same frequency or frequencies a considerable decrease of acuity against the normal of the instrument, the loss of hearing is probaly caused by a disturbance in the conduction mechanism of the ear.

3. If in high frequencies both curves show losses, while in low frequencies losses are registered only by the air-conduction curve, the loss of hearing is caused probably by an obstructive lesion combined by a perceptive factor.

While the rule still holds true, the interposition of the tragus curve between the A. C. and B. C. curves simplifies the original problem and adds many additional informations about the nature of deafness and hearing. In interpreting the tragus test we use the air-conduction and bone-conduction curves as lines of reference and not the audiometric normals of air-conduction and of bone-conduction which are averages, nor a physiologic normal of the tragus conduction which would be an average varying with age.

Using the air-conduction curve as the line of reference we can discover an ossicular ankylosis.

Using for reference the bone-conduction curve, the tragus tests can be classified into types similar to the types of Rinné tests:

Positive (audiograms 1, 2, 4, 5, 8, 9, 10, 11, 12, 15, 18, 23, 27, 29, 35 and 39); diminished positive (audiograms 19 and 20) and absolutely negative (audiograms 16, 17, 31, 33, 34, 37, 39 and 40). Besides that we can distinguish an "increased positive" type in audiograms 3, 12, 24 and 26. Diminished positive tests could be further classified into quantitative sub-groups. But even detailed subdivision and schematization appears to be a rather crude classification because of the many individual features which each audiogram contains. By use of the tragus test we can easily arrive to differential diagnosis between the perceptive, conductive and "mixed" deafness.

No attempt was made to correlate the tragus tests with the Rinné tests. The tuning fork tests have larger observational errors and a different distribution of energy in the conduction apparatus than the audiometric tests, i. e., there are somewhat different casual relations determining the result in the two methods of testing. Under such circumstances, instead of their mutual simple correlation, we should attempt their secondary correlation with the final criteria of clinical and postmortem findings. Microscopic studies of ears compared with their full audiographic records should be very enlightening.

SUMMARY

An audiometric test is described in which the bone-conduction receiver of the 2B Western Electric Audiometer is held against the tragus, impacted into and enclosing the opening of the external auditory meatus. The test is called the "tragus test" and its character is studied on 42 ears. Histories of 21 cases and 46 audiograms are presented. All ears were tested for air conduction, bone conduction and the tragus test. Besides that the audiograms record also the Gellé test which was performed in all frequencies of the audiometer.

The study shows that the tragus curve of normal ears is situated between the air-conduction curve and the bone-conduction curve, and that the patient's age has a similar influence on the tragus curve as on the other two curves. In obstructive lesions the tragus curve is depressed toward the bone-conduction curve.

The level of the tragus curve as related to the level of the bone-conduction curve was found to vary with the condition of the middle ear mucosa. The osseous block in the oval window reduced the hearing in the tragus test more than a fibrous fixation of the stapes. A theory was expressed, according to which, the

threshold vibrations in the tragus test reach the annular ligament through the membranous lining of the middle ear and the tragus conduction was designated as having a membranous character.

A group of ears was studied which in low tones showed crossing of the air-conduction curve below the tragus curve. This tragus-air-curve crossing was interpreted as ankylosis of the ossicular joints. Attention is drawn to frequent nasopharyngeal involvement in cases of catarrhal deafness and to the similarity of the tragus test with the Rinné test.

30-28 THIRTY-SIXTH STREET.

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Clinical Notes

LXXIX

A STUDY OF TEN CASES OF DEEP INFECTION IN THE NECK

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The anxiety caused by the loss of a patient, whether due to poor judgment, ignorance or mismanagement is difficult to forget. The repetition of such mistakes cannot be born without worry. The stimulus for more knowledge brought on by such incidents has frequently taken me back to my records, to the dissecting room and to the literature, in a search for more definite information.

Clinical cases of this nature have incited me to make a more intensive study of the deeper infections in and about the fascial sheaths of the neck. Local symptoms and signs may be explained by applied anatomy, and where correct analysis can be made on these grounds, the site can be located and the proper remedy given. Deep infections in the neck do not respect age, sex or station in life. Wherever disease of the upper air passage is prevalent, this complication is likely to follow.

The innocent tonsillectomy, no matter how well done, nor how correct the technique may be, often results in abscess, thrombophlebitis and meningitis. During the course of acute tonsillitis one can well be apprehensive of deeper adjacent structures becoming involved until such time as the patient has entirely recovered. There are no more dreaded or dangerous complications than thrombophlebitis of the veins and venous sinuses of the neck and head following throat infections. The inflammation in the throat may be severe, and reveal frank pus, or it may be mild and almost escape notice—yet; a septic temperature may arise with thrombosis, and meningitis as an aftermath. In spite of the voluminous literature during the past eight years, and the intensive anatomical investigations, cases still go on to fatal terminations. This is not only due to an oversight by the otolaryngologist, but the general surgeon as well. Patients two and three of this report each suffered with an abscess in

the retropharyngeal spaces, and while they did not have primary tonsillitis, their tonsils with other structures were red and infected. Case eight was due to trauma, but all the others had a definite infection in or about the tonsils, or had recently undergone a tonsillectomy, and most of these had occurred when the tonsils had been removed after the use of local anesthesia.

Shapiro[1] found that out of one hundred and ten cases reported in the literature, in ten the anesthetic was not definitely stated, but of the remaining one hundred, ninety-four had had a local anesthetic. He cites three possible sources of infection in local anesthesia. (1) Injection through the tonsil tissues carrying organisms from the infected tonsil itself. (2) Contamination of the needle or the solution prior to injection, and (3) infection of the mucous membrane at the site of injection. Three of my cases had had a local tonsillectomy, one was done by myself, and the other two by physicians in other communities.

There is also another type in which the fascial spaces are the seat of the initial invasion and for which throat surgery is not responsible. In a study reported by A. L. Beck,[2] he thinks the tonsil and the pharynx are the most common causes. This was true in cases four and seven of this report. Other sources are the adenoid, pharyngeal lymph nodes, mastoid, petrosa and the nose and sinuses. Case one of this series followed an infection of the sinuses and adenoids.

Those cases, not due to surgery, usually follow some upper respiratory infection and are probably caused via the lymph nodes and vessels. Surgical interference is indicated early and if not resorted to, many serious complications may take place.

An intimate knowledge of the anatomy in this area is essential to proper management of deep infections in the neck. This part I shall omit and for those who are interested, I will refer them to the excellent anatomical studies of such investigators as Mosher,[3] Hall,[4] Beck,[2] Furstenberg,[6] Barnhill[7] and Davis.[8]

REPORT OF CASES

CASE 1.—Baby T., a nine-months-old male infant, entered the hospital on February 8, 1937. The main complaint was a fever, loss of appetite, inability to open the mouth, difficulty in swallowing, and a swelling in the left mandibular region. At that time he had a temperature of 102 degrees. The white blood count was 16,000, the left cervical region was swollen, a firm mass which extended under the angle of the jaw could be palpated over the left

mandible. This was extremely tender. The nostrils were congested. The turbinals were red and swollen and there was pus in both nostrils. Palpable glands were present on both sides of the neck. There was much difficulty in getting the mouth open. The tonsils were normal in size but somewhat reddened and the left was pushed toward the median line. The pharyngeal membrane was red, thick and some mucopurulent secretion covered its surface. Other physical findings were negative or irrelevant.

On February 9, the second day after admission, the swelling about the jaw increased in size and the jaw was firmly set. Due to this extreme spasm I gave the child a few whiffs of chloroform and discovered definitely a swollen mass pushing the tonsil further toward the median line. I could feel no fluctuating point and hence waited for further developments. The following day, under ether anesthesia, an incision was made in the posterior pillar and with curved forceps the mass was gently opened. This was followed by quantities of pus (a culture showed hemolytic streptococcus). The wound was enlarged, the cavity aspirated. The child improved rapidly. The temperature dropped from 103 to 100 degrees, the mouth could be opened and the child improved in general. This continued for two days, when the temperature again shot upward. Trismus appeared once more and following this a rather severe hemorrhage took place. The child vomited quantities of dark blood. About six hours later it was striken by a fulminating erysipelas, which covered the entire face and neck in a very short time. Erysipelas anti-toxin was administered. Another hemorrhage took place, a blood transfusion was given, but the following day the child died of general sepsis.

Comment: This child had a parapharyngeal abscess with all the cardinal symptoms. It is now my judgment that two mistakes were made in the treatment of this case: (1) I waited too long before incising the abscess. (2) It should have been opened externally instead of through the mouth. The internal incision did not promote adequate drainage and the long wait caused destruction of the great vessels in the neck, causing a debilitating hemorrhage. Erysilepas contributed to the violent ending.

CASE 2.—Baby H., one year of age, male, was brought to me on April 5, 1934. It was having labored breathing, with a slight inspiratory tug, a large swelling on the right side of the neck and high fever. This was accompanied by an almost complete inability to swallow.

The child had suffered with a cold two weeks previous and this was followed by swelling in the right side of the neck. The difficulty in swallowing, the high temperature and the respiratory embarrassment had been getting worse for two days.

Under general anesthesia, a large retropharyngeal abscess was incised on the right side and quantities of pus aspirated. An obstructive mass of infected adenoid tissue was removed. I next incised a broken-down abscess under the superficial fascia in the right anterior cervical region.

The child made an uneventful recovery and left the hospital in four days.

CASE 3.—Baby G., an infant male, age eight months, was referred by the pediatrician after he had made a diagnosis of retropharyngeal abscess. When seen by me on May 29, 1934, he was suffering from difficult breathing, high fever, a loss of weight and an inability to swallow food. He had been ill for about five weeks, following influenza.

A large fluctuating mass extended deep down the pharyngeal membrane toward the right side of the larynx compressing it enough to give him noisy breathing.

The abscess was incised and great quantities of pus followed. He made an uneventful recovery and left the hospital three days later.

CASE 4.—Miss A. G., a white girl, age 13, complained of sore throat, stiff neck, swollen, tender glands, extreme dysphagia, chilly sensations and temperature. These symptoms had gradually increased in intensity for one week, at the end of which time she entered the hospital. She had had her tonsils removed one year before, and suffered with severe colds and periodic enlargement of the cervical glands since having measles four years before.

On admission her temperature was 103.2 degrees, her nostrils were filled with pus and the pharyngeal membrane was fiery red. There was a definite swelling in front of and about a tonsil stump at the superior pole of the left fossa. Hemolytic streptococcus was found after culturing the pharyngeal membrane. There was a marked swelling and tenderness in the left anterior cervical triangle. The patient was pale and extremely septic. Trismus was present but not so marked but what a good inspection of the throat could be made. With topical anesthesia I made an incision high through

the anterior pillar. To my surprise the patient suffered no pain. There seemed to be almost complete anesthesia on the affected side. The opposite side was normal. Rather free bleeding took place but no definite pus could be seen. The following morning it was noted that the eye on the left side was somewhat fixed in all directions with complete paralysis of the external rectus and that the left pupil was dilated. She expectorated some blood at times, but no pus could be washed from the incision. The temperature continued to rise, the eye became fixed, with some swelling of the upper lid, and beginning proptosis. At the end of twenty-four hours, she suddenly died. No autopsy was permitted.

Comment: This, in my judgment, was a case of cavernous sinus thrombosis, induced by a deep infection in the neck, fulminating in type.

The dilated pupil was due to a stimulation of the sympathetic fibers that are given off by the superior cervical ganglion and encroached upon when infection reached the parapharyngeal space.

CASE 5.—On April 12, 1923, with local anesthesia, I removed the tonsils of Miss F. F., a school teacher, age 30. Eight days following, the right side of the neck over the angle of the jaw began to swell. This continued to increase in size until April 24, when I inserted an artery forcep into a bulging tonsil fossa. This was followed by a free profuse flow of pus. Eleven days later the condition had entirely cleared up and the patient seemed well again.

Comment: Parapharyngeal abscesses respond more quickly and with less care by puncturing the superior constrictor muscle if the tonsil fossa bulges. The external route will prove more satisfactory when the tonsil remains in situ.

CASE 6.—Mr. B., a salesman, age 22, complained of chills, temperature, inability to open the mouth and a large, tender mass in the region of the left mandible.

He gave the history of having had a tonsillectomy with local anesthetic two weeks before. Six hours after the tonsillectomy, he had a severe hemorrhage from the left side. He returned to his physician and after some manipulation the vessel was grasped and ligated and the hemorrhage stopped. This was followed by considerable pain and distress on the left side of his throat. Three days following, pain, swelling and difficulty in opening the mouth, began to increase in intensity. In this condition he came to my office on September 8, 1937. It was difficult to open the mouth but one

could see the faucial region pushed toward the median line. On the outside the swelling extended from the parotid gland over the mandible and covered the entire sublingual area. He was admitted to the hospital with a temperature of 103 degrees, followed shortly by a chill lasting fifteen minutes. He had a white blood count of 15,000 with 91 per cent segmented cells. He was put to bed, given five cc. of prontosil intramuscularly every four hours. He began to improve slowly during the 9th, 10th and 11th, and on the morning of the 12th the abscess ruptured internally, followed by the expectoration of great quantities of pus. He then began to improve with an abrupt fall in all his symptoms and left the hospital on the 14th, entirely well.

Comment: This patient had a definite infection in the parapharyngeal space following tonsillectomy after the use of a local anesthetic. Prontosil was administered and his symptoms were held in abeyance. Fortunately, the abscess ruptured without complication. If I had a similar case today, I would incise the abscess externally and give the prontosil following.

CASE 7.—Mr. J., school teacher, age 24, married, was admitted to the hospital on February 16, 1938. He complained of an almost total inability to open the mouth, painful deglutition, hoarseness, chills, temperature, and a large swollen mass about the left jaw, and the left side of his face.

He gave the history of having been ill for one week. His family physician had made a diagnosis and treated him for an acute tonsillitis until the present symptoms began. His past history was negative. He had had no severe attacks of tonsillitis before.

It was with difficulty that any inspection could be made in the mouth. However, it could be seen that the left tonsil and the surrounding tissues were pushed well over the median line. His temperature on admission was 101 degrees, which quickly rose on the following morning to 104 degrees. His white blood count was 42,000 and his segmented cells 81 per cent. On the day following his admission, a local anesthetic was administered, a large incision was made under the angle of the jaw (after Mosher's method). With blunt dissection the abscess cavity was soon entered and great quantities of pus escaped. From that time until he left the hospital, two weeks later, he was not in danger at any time. He soon returned to his work and has been well ever since.

Comment: The abscess was so extensive and deep that his larynx was beginning to be involved, and I feel that if the external

route had not been taken this patient would probably have met with a fatal result.

CASE 8.—Mr. O. W., age 36, on January 24, 1939, was hit in the left jaw by an assailant. Two days later the jaw began to swell, and two days following this a physician was called because of an inability to swallow without a return of the food through the nostril. On this day he had a severe chill, followed by a high temperature. On February 3, he entered the hospital with a temperature of 104 degrees. There was a marked swelling over the left jaw extending into the left cervical region and a decided bulging of the faucial area, which displaced the uvula past the median line and caused great difficulty in swallowing. He had caries of the teeth with an extensive pyorrhea. The x-ray revealed an abscess on the left second molar accompanied by a commuted fracture of the mandible which directly connected with the abscess of the tooth. His white blood count was 12,500, the segmented cells 79 per cent. There was a trace of albumin in the urine. Under medical care; irrigations in the mouth, steam, external heat, the swelling soon began to recede. The bulging of the tonsil and tonsil area receded to normal but the jaw remained swollen. This gradually decreased in size. The infected tooth was extracted and pus flowed freely into the oral cavity from the socket. An external incision was made and quantities of pus evacuated. He is making an uneventful recovery.

Comment: Trauma caused the fracture of the jaw, which unfortunately connected with an alveolar abscess. Just why the peritonsillar infection took place is probably explained by a rupture of the submaxillary faucial space into the anterior compartment of the pterygomaxillary fossa. This could easily be caused by trauma or infection.

CASE 9.—Mr. S., male, age 39, entered the hospital February 28, 1927. He complained of obstructed nasal breathing, a purulent discharge from the nostrils, headache, sore throat, fullness and aching in the ears. He gave a history of having had sinus infection since the pandemic of influenza in 1918. His present trouble dated back about two weeks.

He had an infection in both maxillary sinuses, a culture from which gave a positive growth of hemolytic streptococcus.

He improved uneventfully for about ten days, under strict medical care, and then developed a septic temperature accompanied by sore throat on the left side and swelling of the left cheek.

An abscess formed in the buccal region which was incised perorally. The patient made a slow recovery from the air passage infections, under prolonged medical care. An acute nephritis developed from which he still suffers.

CASE 10.—Mrs. M., age 42, housewife, was admitted to the hospital on October 18, 1937. She complained of severe unrelenting headache, soreness in the left side of the neck, and an inability to move the head without pain in this region. She had a high temperature and was extremely ill.

Her past history was negative other than having a few joint and muscle pains at times for which she consulted her family physician. On October 12, he removed her tonsils under local anesthesia. The right one was removed first without difficulty, but when he started to remove the left one, she complained of some distress, at which time he reinserted the needle and gave more novocain; at this time the patient had a sudden severe pain. The tonsil was removed and she was assured that the pain would discontine. After she returned home this increased with more severity, so much so that the doctor was called and morphine given. A quarter of a grain of morphine was needed every four hours until her admission to the hospital. Two days before being admitted, headache had been severe and constant.

There was no difficulty in opening the mouth. The tonsils had been cleanly removed and the fossæ were as normal as one would expect to find on the sixth day after tonsillectomy. There was probably a little more hyperemia about the left side. The nose, the nasopharynx and the ears were all negative. No swelling and no glandular enlargement could be detected on either side of the neck. There was tenderness on pressure in this area, along the course of the carotid artery and jugular vein, and when she attempted voluntary movements of the neck, the pain was severe.

She lay with her head motionless, as if her cervical spine were injured. The pupils were regular and reacted to light; the media clear, the fundus and nerve head normal. A lumbar puncture was done, the fluid was clear and under slightly increased pressure. The cell count was 166, otherwise negative. Hemoglobin, 80 per cent; coagulation time, three minutes; erythrocytes, 4,640,000; white blood count, 12,700. The differential white count showed small lymphs, 2 per cent; large lymphs, 1 per cent; eosinophiles, 1 per cent, and segmented cells, 96 per cent. On October 21, another spinal puncture was done, with the fluid clear, the pressure normal and the cell

count 70 per cent. The Pandy was negative, no organisms found in cultured or centrifuged, strained spinal fluid. The blood culture was negative, taken on two different occasions. On the day following admission, the internist reported a positive Babinski, a positive Kernig, and suggested four possible diagnoses: (1) Meningitis. (2) Septicemia. (3) Ascending infection in the neck. (4) Encephalitis. On admission her temperature was 101 degrees and the pulse rate 125. The temperature rose to 104 degrees and ranged from 103 to 107 degrees. Her pulse was never lower than 100 and usually kept about 130 beats. She was extremely ill and seemed to be suffering from intracranial complications. On the morning of October 21, a notation was made on her record as follows: "She appears to have an acute encephalitis following a local tonsillectomy. The spinal fluid is clear with 70 cells, no protein, pupils dilated and the neck still rigid. Reflexes: Kernig, Babinski, Oppenheim, Gordon and Brudzinski, all positive." Her temperature went to more than 107 degrees, and on October 22 she died.

Post Mortem: The skull cap was removed, the dura appeared normal, not only over the vault, but over the base as well. It was my own opinion that she had died of encephalitis or a brain abscess, therefore the entire brain was removed and sent to the pathologist without further investigation of the base of the skull, the vascular sinuses or the carotid region. After the pathologist had sectioned the brain and did a histologic examination of suspected areas, he pronounced it normal.

Comment: All of the symptoms above enumerated came on suddenly and dramatically following a tonsillectomy after the injection of novocain about the tonsil. I am at a loss to know the cause of her death. I feel sure she died a cerebral death. If I had investigated the petrosal, the circular, the cavernous sinuses, there is a possibility the trouble could have been found. The carotid sheath and its coverings through the skull should also have been investigated, and yet, it seems that if infection had passed by this route, something should have been found in the cranial cavity. I cite this case for the problem it presents. Certainly the infective agent must have entered through the deeper structure of the neck at the time the tonsils were removed.

Frank and Scheer[5] investigated the route of intracranial complications from parapharyngeal infections. They say under pathways of infection that the spread from the buccopharyngeal region to the endocranium as described in the literature is by venous chan-

TABLE I

CASES Age and Sex	ETIOLOGY Focus of Infection	ANATOMIC LOCATION OF PATHOLOGY Para-Pharyngeal Space	Pre-Vertebral Fascia	Bucco-Pharyngeal Pretracheal	Carotid Sheath	Lymph-adenitis	COMPLICATIONS	TREATMENT RECEIVED	REMARKS
No. 1 Baby T. 9 mo., M.	Nasopharyngitis Tonsillitis	Lateral pharyngeal abscess	....	....	....	Secondary	Hemorrhage Erysipelas	Peroral incision Transfusion Erysipelas Antitoxin	WBC 16,000 Hemolytic streptococcus Death
No. 2 Baby H. 1 yr., M.	Infected adenoid Nasopharyngitis Sinusitis	....	Retro-pharyngeal abscess	....	....	Superficial lymph-adenitis	Suppurating lymphadenitis	Peroral incision Drainage of superficial adenitis	Recovery
No. 3 Baby C. 8 mo., M.	Infected adenoid Nasopharyngitis	....	Retro-pharyngeal abscess	....	....	....	None	Peroral incision Aspiration	Recovery
No. 4 A. C. 13 yrs., F.	Nasopharyngitis Infected tonsil stump Influenza	....	....	....	Ascending infection	Secondary	Cavernous sinus thrombosis Hemorrhage	Peroral incision of peritonsillar abscess	WBC 12,000 Death
No. 5 F. F. 30 yrs., F.	Tonsillectomy (local)	Lateral pharyngeal abscess	....	....	....	Secondary	None	Incision through tonsil fossa	Recovery after 11 days
No. 6 B. B. 22 yrs., M.	Tonsillectomy (local)	Lateral pharyngeal abscess	....	....	....	....	None	Incision through tonsil fossa	Recovery after 4 days
No. 7 W. J. 24 yrs., M.	Acute tonsillitis	Lateral pharyngeal abscess	....	....	....	....	None	External incision	Recovery after 15 days
No. 8 O. W. 36 yrs., M.	Alveolar abscess Trauma Fractured mandible	Lateral pharyngeal abscess	....	....	....	....	Fractured mandible	Extraction of tooth Supportive	Spontaneous oral drainage now being treated
No. 9 P. S. 39 yrs., M.	Pansinusitis Pharyngitis Infected tonsils	....	....	Bucco-pharyngeal abscess	....	Secondary	Acute nephritis	Peroral incision	Slow recovery Tonsillectomy later
No. 10 M. McL. 42 yrs., F.	Tonsillectomy (local)	....	....	....	Carotid sheath infection	....	....	Supportive	Death (cause unknown)

nels, by direct extension of a phlegmonous inflammation from the parapharyngeal space, by erosion of bone and by the lymphatics. They experimented on dogs by injecting living, virulent organisms into the carotid sheath to ascertain if a direct pathway could be demonstrated along the sheath of the internal carotid artery to the cranial cavity. "In two instances, organisms were encountered in the sheath of petrous and cavernous portions of the internal carotid artery."

In all the other dogs experimented on (28 adults, 19 puppies), no organisms could be recovered from the brain or its coverings.

In the light of these experiments, it would seem that the infection in this case did not travel by way of the sheath of the artery. However, if infection was injected directly into the artery, encephalitis could have resulted and the pain could have originated from an infection of the sheath about the site of injection.

CONCLUSIONS

An analysis of ten reported cases suggests the following:

1. That many cases of deep infections in the neck, if treated early, can be treated conservatively.

2. That peroral incision will prove satisfactory in retropharyngeal abscess.

3. That the external incision should be resorted to in all cases of infection of the parapharyngeal space where trismus and swelling are present and where tonsillectomy has not been performed. The incision may be made through the fossa when the tonsils have been removed and the tonsil bulges.

4. That hemorrhage may take place from an internal incision if prompt free drainage is not obtained.

5. That most of the deep abscesses of the neck result from infection in and about the tonsil or follow tonsillectomy, particularly when local anesthesia is used.

6. That probably case ten died from an encephalitis by bacteria being injected directly into the carotid artery, even though no evidence could be found at postmortem examination.

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LXXX

REPORT ON TWO CASES OF CARCINOMA OF THE SPHENOID AND ETHMOIDS

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In presenting two cases of extensive carcinoma of the sinuses and adjacent tissues, I do so because they exemplify:

1. The necessity of a complete history and not just an ear, nose and throat inquiry.

2. The advisability of correlation of history, the symptoms, physical findings, laboratory findings and x-ray.

3. When giving x-ray treatment for relief of pain in what appears to be a hopeless case, give it in the dosage that you would in expecting a cure.

REPORT OF CASES

CASE 1.—The first case is that of a patient, Mrs. W. S., age 66 years. She was referred to me by an associate, Dr. F. H. Haessler. She complained of a constant left-sided headache, particularly in and around the left eye. This had come on following a bad cold six months before. The patient stated that she always had a certain amount of sinus infection and had been under treatment for it. At the time of examination, she stated that there was very little discharge from the nose.

Dr. Haessler's report on the eyes is as follows:

"When Mrs. S. was first seen in 1933, her vision was 20/20 in each eye with correcting lenses. In February, 1937, she was seen again with normal vision with correcting lenses in the right eye and barely light perception in the left. Vision in the left was not sufficient to get a field. Fundus in both eyes were normal. In July there was an exophthalmus of 2 mm. in the left eye from the orbital rim. There was puffiness of the left lids. Further consultation upon the ear, nose and throat was insisted upon, and the patient was referred to Dr. Tolan."

My examination was as follows: The patient gave the picture of a person suffering with excruciating pain. The temperature and pulse were normal. Mouth: small, buried tonsils; complete upper and lower dentures. No postnasal discharge. Nose: left side of the nose was edematous with some watery discharge, and on shrinking, considerable pus was seen in the middle meatus. Just below the tip of the middle turbinate was a growth about 1 x 2 centimeters which came from the lateral wall. On probing this, it was found to bleed readily. Ears were essentially negative. Transillumination of the sinuses showed the frontal and antrum on the left side to be black in contrast to the opposite side.

The patient was advised to have the growth removed for biopsy. Suggestion was made as to treatment of the sinus infection and an x-ray of the sinuses was ordered.

A biopsy was done at the time of the first examination, July 1, 1937, and was examined by Dr. Enzer, pathologist, who found:

"Microscopic section discloses it to be occupied by a rather anaplastic epithelial growth of the transitional cell type, invading the tissue, forming large medullary nests and cords. There is a considerable amount of acute inflammatory reaction. The stroma is moderately fibrosed surrounding the tumor, but the intercellular stroma is scanty. The tumor is highly malignant.

"Diagnosis: Transitional cell carcinoma.

"Comment: The origin of this tumor could be the nasal pharynx, antrum or nares."

X-ray examination done by Dr. Morton on July 12, 1937, reported:

"There is a very marked destruction of the posterior part of the roof of the orbit on the left side and a destruction particularly of the lesser wing of the sphenoid and left anterior clinoid process. In the lateral view some destruction of the sphenoid ridge of the left parietal bone can be demonstrated. There is, in addition, a slight infiltration of the left frontal sinus and maxillary sinus. There is some destruction also in the left sella turcica involving the anterior clinoid process and displacing the posterior clinoid process backwards. The floor of the sella is also damaged and there is some infiltration in the left sphenoidal sinus.

"Diagnosis: Malignant tumor situated in the anterior part of the middle fossa on the left side involving rather extensively the contiguous structures."

In view of the microscopic findings and the x-ray picture, a very gloomy prognosis was given to the relatives. We advised, however, that x-ray treatments should be given with the thought of alleviating the headaches. This was consented to and the treatments started on July 19, 1937, by Dr. Morton. We felt, however, that it would be advisable to treat the patient with heavy doses.

Dr. Morton reported:

"Treatment was given through three fields: one over the left eye, one downwards in the left frontal parietal area, and one directed inwards on the lateral aspect of the head, the center of the field being about midway between the external canthus and the external ear; 10 x 10 centimeter fields were used. Treatment was given every second day and three fields treated on each occasion. The dosage each time was 180 r. The x-ray factors were 200 K.V.P.: Thoreaus filter; 50 cms. distance. Treatment was completed on August 11 (twenty-three days).

"A few days following the conclusion of the treatment, considerable reaction appeared in the temporal region. This grew worse until an area four or five centimeters in diameter was severely involved. Complete epilation of the anterior quadrant of the scalp occurred. The skin was treated with Dodd's lotion. Her general condition was rather poor. General measures were used to combat an anemia. By September 8, 1937, a very definite improvement had taken place and the wound was healing nicely. By November 8, 1937, all the skin had healed and was soft and pliable. General condition was also greatly improved. Severe headaches and pain in the eye have entirely disappeared.

"In February, 1938, the x-ray of the skull and sinuses was repeated and considerable improvement noticed. The extensive destruction was still present but was definitely no worse than before. The edges of the eroded area which were previously rough and irregular are now limited by good cortical bone. The bone appearance by x-ray seems definitely quiescent, while before it looked extremely active."

The x-ray findings on December 19, 1938, showed no evidence of recurrence; on the contrary, the bone looked definitely better.

The patient's headaches increased considerably during the first two weeks of treatment. From that time on, they gradually decreased until a month following the end of the treatment, when only occasionally were they severe enough to take salicylates, which relieved them. Within two months of the beginning of the x-ray treatment, the patient was free from headaches and has been so ever since except for occasional headaches which she attributed to other causes. Skin at that time was soft and pliable and not at all unsightly, and the hair on the scalp had come back nicely. Her general condition was excellent.

Dr. Morton's comment:

"This dosage that was given for a person of her age was quite large. In a person twenty years younger it would not have caused as severe a reaction. No further x-ray is indicated at this time, although if the condition recurs, it will be possible to radiate her again."

We, of course, are not losing sight of the fact that only a year and one-half has passed since treatment was completed and appreciate that recurrences are still very possible, but are encouraged with not only the patient's general condition and lack of symptoms but also with the appearance of the bone x-ray.

CASE 2.—The second case is that of a patient, Mr. A. K., age 67 years. The patient was first seen by me on November 9, 1938, at which time he complained of constant headache above the right eye for three months. He had been under observation and treatment for sinus infection during that time. The pain was particularly aggravated by blowing the nose and was constant throughout the day. At the time they started, he got relief with salicylates, but in the last three weeks nothing helped. In the past two weeks, he had been seeing double. He stated that his general health was good. Examination of the ear, nose and throat was essentially negative. Transillumination of the sinuses was normal. There was no postnasal discharge, nor was there any obstruction or discharge in the nose.

The patient was referred to his eye man, Dr. Warner, who reported paresis of the external rectus. Otherwise, the eye examination was negative except for slight contraction of the fields on the temporal sides. The eye was covered for awhile but the patient experienced no relief. He was seen again by me on November 17,

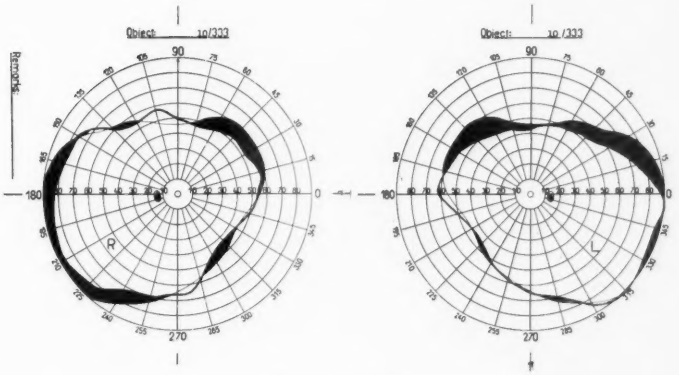

Fig. 1. Case 2.

1938, and x-ray was advised. He was x-rayed by Dr. Morton, who reported:

"The frontal and maxillary sinuses are radiographically normal. The ethmoid sinuses are well visualized both in their anterior and posterior portions. They are also normal.

"There is a marked destruction of the floor of the sella and posterior clinoid processes. The destructive process apparently invades the greater portion of the sphenoid sinuses. The appearance is that of a tumor arising in the sella turcica or sphenoid sinuses.

"The calvarium is normal. The appearance suggests more a primary than a metastatic tumor.

"Diagnosis: Extensive tumor involving the sella and sphenoid sinuses."

At this time, however, going further into the history, it was learned that he had had a rectal operation on January 3, 1938, and there was removed a grade 2 adeno-carcinoma of the rectum. The patient made a very good recovery at the time of operation.

On December 1, 1938, under local anesthesia, the anterior wall of the sphenoid was removed on the left side. There was no pus in the sinus, but a boggy red mass was seen. A small specimen was removed for examination. It was examined by Dr. Hansmann, pathologist, who stated:

"Microscopic examination revealed a mucous secreting adenomatous tumor with a marked reproduction of gland in a fairly dense stroma with some lymphoid reaction. The tumor seemed to be coming from bone and was pushing a ciliated columnar epithelium ahead of it. The appearance was that of a low-grade malignancy:

"Interpretation: Although the naming of tumors in metastatic lesions is not accurate, this tumor has the histological features of large bowel. Tumors from sphenoid sinuses are usually pseudostratified and not well developed. The fact that this has evidently arisen in bone and pushed sphenoid epithelium ahead is indicative. On these facts, the tumor is considered metastatic, and since there has been a carcinoma of the rectum removed earlier, it is most likely metastatic and of that source."

I feel that if a thorough history had been taken on this patient at the time of his first examination, we might have discovered this condition a bit sooner. Following the biopsy, the patient's headaches were increased, and having in mind the case just presented, we decided to give him some x-ray. However, a week following the biopsy, the patient became quite yellow, extremely ill, and from the signs and symptoms, his medical man felt that metastasis to the liver and lungs had occurred. He died from a complicated pneumonia on January 22, 1939.

The unusual site for metastasis prompted me to report this case.

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The Scientific Papers of the American Bronchoscopic Society

LXXXI

CERTAIN PHASES OF RESPIRATORY FAILURE: AN EXPERIMENTAL STUDY OF SUBACUTE OBSTRUCTIVE ASPHYXIA*†

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There are many classifications of asphyxia. Meakins and Davies[1] divide them into seven etiologic groups. The forms which concern the laryngologist are mainly obstructive, and that type which is produced by the ill effects of anesthetic gases acting upon the respiratory center. My remarks shall be limited to the former type only. The chief factors controlling respiration are of nervous and chemical origin. According to McLeod,[2] the vagus plays a most important role in the control of respiration, for electrical experiments indicate that the vagus contains two kinds of afferent fibers to the respiratory center, one kind stimulating inspiration and the other stimulating expiration. This phenomenon is known as the Hering-Breuer reflex. Various experiments indicate that the vagus exerts an influence on the depth of respiration, but not on its rate.

The chemical control of respiration is concerned chiefly with three factors, i. e., the arterial carbon dioxide tension, the arterial oxygen content and the hydrogen ion concentration designated by the symbol pH. The mechanism of respiration is not entirely understood and death from its failure in many instances may involve one or more related factors. The thoracic pressure, Hering-Breuer reflex, carotid body reflex, cerebral blood flow and the gaseous changes in

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the blood are so inter-related that it may be difficult to ascribe death of an asphyxial nature to an independent phenomenon.

A review of the literature reveals that a great deal of work has been done on many types of asphyxia, though there is little to indicate that much has been written about chemical changes in obstructive asphyxia. Caryllos[3] acutely obstructed the trachea of eighteen dogs in order to determine the comparative effects of certain resuscitating measures, and Eastman[4] has recently induced acute asphyxia in dogs, with nitrous oxide in order to evaluate the resuscitating properties of carbon dioxide mixtures, and that of oxygen alone.

The purpose of this paper is to discuss, in a limited way, some of the chemical changes in the arterial blood, which are associated with subacute obstructive asphyxia and its effect upon life under certain circumstances. It is generally known that when an animal or human is subjected to certain phases of asphyxia, carbon dioxide is excessively accumulated in the arterial blood, which acts as a respiratory stimulant. If this asphyxial condition is suddenly relieved, as it may be in certain instances, the carbon dioxide is blown off in excess by the overventilation of the lungs and the arterial blood rendered more alkaline. In other words, the acid-base ratio of the blood is acutely upset.

In order to explain the cause of certain deaths which have occurred immediately following a successful tracheotomy for the relief of obstructive asphyxia, some authors have adopted these findings. Negus[5] states that some of these patients may cease breathing immediately after placing the cannula in the trachea, while others may not die so soon. He explains this phenomenon on the following biochemical basis, stating in substance that when asphyxia is caused by laryngeal obstruction the arterial carbon dioxide is raised above its normal percentage in the form of carbonic acid. This, in turn, sets the respiratory center at a higher acid-functioning level, which may be proportional to the degree of asphyxia. In advanced asphyxia the stimulating level is quite high and when the airway is suddenly and completely opened, breathing becomes immediately deeper and faster. This excessive breathing causes a wash-out of carbonic acid, resulting in an increased alkalinity of the blood, which now bathes the respiratory center with a depressing and sometimes fatal effect.

The following case is of interest because: (1) death occurring under similar circumstances is very unusual; (2) the clinical features of this death parallel other cases reported as acapnea; (3) the explanation that has been offered for this form of death may be hypothetical.

CASE REPORT

CASE 1.—G. S., male, age 21, was admitted to the hospital November 1, 1936, ill with Ludwig's angina. His fever on admission was 104 degrees, pulse 128, respiration 32 and labored. I saw this patient at four o'clock in the afternoon, at which time dyspnea was urgent. Tracheotomy was advised and immediately performed, using novocaine anesthesia with no unusual difficulties. When the trachea was opened the surrounding area was reasonably dry and the cannula was inserted with the usual sense of satisfaction. The patient breathed eight or ten times through the unobstructed airway and then died. Permission for necropsy was not obtained. While this case simulates others which have been reported as "carbon dioxide deficiency death," it would seem just as reasonable to ascribe this mechanism to a possible cerebral damage resulting from anoxia.

The etiologic analysis of such an unusual death is not easy, because it is difficult to find in the literature similar case reports, with necropsies, and secondly, because there are no parallel animal experiments found to justify the explanation of this form of death. In view of this fact, certain experimental studies were undertaken upon dogs in order to determine: (1) whether death could be induced by subjecting animals to a similar process which may have caused death in humans and, (2) if death could be and was induced by these experiments, what degree of change occurred in the total arterial CO_2 and the pH.

In order to duplicate as nearly as possible the same mechanism in the animal as that which is assumed to cause a similar condition in the human, tracheotomies were done on 42 animals, using local anesthesia. A suitable cannula was used so that the tracheal lumen of each dog was slightly distended in order to prevent leakage of air around the tube. In one group the period of obstruction existed for twenty-four hours in order not only to accumulate carbon dioxide in the blood but to allow sufficient time for the body tissue to saturate and permit a compensatory increase in blood bicarbonate. This was accomplished by obstructing the tube at the outset about 75 or more per cent, and during the latter portion of the twenty-four hour period the lumen was further restricted until urgent dyspnea was obtained, and at the point of collecting the first blood an oxygen crisis or its approach was precipitated. Fifteen cc. of blood were drawn immediately from the femoral artery for the first CO_2 estimate and pH determination. Having completed this, and while the animal was still in the apneic stage of asphyxia, the plug in the tracheal tube was suddenly released and the animal given an oppor-

Dog	Obst. Period	First pH	Second pH	Diff.	First CO_2	Second CO_2	Diff.	Approx. Interval
1	24	7.48	7.56	0.08	46.64	41.47	5.17	6 min.
2	24	7.31	7.39	0.08	26.97	25.97	1.00	6 min.
3	24	7.37	7.59	0.22	45.54	44.36	1.18	6 min.
4	24	7.08	7.22	0.14	45.75	40.81	4.94	6 min.
5	24	7.16	7.16	0.00	43.25	42.90	0.35	6 min.
6	24	7.52	7.58	0.06	46.20	45.31	0.89	6 min.
7	24	7.37	7.62	0.25	47.12	41.27	5.85	6 min.
8	24	7.49	7.50	0.01	40.06	36.37	3.69	6 min.
9	24	7.40	7.48	0.08	43.71	43.30	0.41	6 min.
10	24	7.36	7.39	0.03	47.28	43.52	3.76	6 min.
11	24	7.11	7.41	0.30	46.54	42.70	3.84	6 min.
12	24	7.36	7.36	0.00	45.39	44.60	0.79	6 min.
13	24	7.02	7.27	0.25	42.99	30.96	12.03	6 min.
14	24	7.35	7.53	0.18	52.01	44.77	7.24	6 min.
15	24	7.43	7.44	0.01	56.08	45.67	10.39	6 min.
16	24	7.46	7.48	0.02	52.98	52.33	0.65	6 min.
17	24	7.37	7.45	0.08	59.37	44.59	14.78	6 min.
			Average	.10			4.52	
18	24	7.24	7.51	0.27	59.39	48.69	10.70	3 min.
19	24	7.39	7.48	0.09	43.90	34.96	8.94	3 min.
20	24	7.54	7.80	0.26	47.80	33.82	13.98	3 min.
21	24	7.79	7.62	(0.17)	40.58	38.47	2.11	3 min.
22	24	7.16	7.39	0.23	42.87	37.62	5.25	3 min.
23	24	7.32	7.65	0.33	47.54	36.29	11.25	3 min.
24	24	7.32	7.32	0.00	44.03	25.95	18.08	3 min.
25	24	7.46	7.50	0.04	44.73	44.35	0.38	10 min.
26	24	7.32	7.49	0.17	49.18	42.59	6.59	10 min.
27	24	7.33	7.49	0.16	42.72	39.42	3.30	10 min.
28	24	7.37	7.51	0.14	44.35	40.03	4.32	15 min.
29	24	7.32	7.44	0.12	38.44	33.65	4.79	15 min.
30	24	7.48	7.85	0.37	35.77	31.35	4.42	20 min.
			Average	.18			7.23	
31	15	6.88	7.28	0.40	54.74	39.65	15.09	6 min.
32	15	7.21	(7.11)	0.10	44.58	25.10	19.48	6 min.
33	15	7.31	7.40	0.09	47.58	46.12	1.46	6 min.
34	15	7.31	7.39	0.08	46.71	41.92	4.79	6 min.
35	15	7.21	7.30	0.09	51.23	50.80	0.43	6 min.
36	15	7.40	7.43	0.03	45.71	33.96	11.75	6 min.
37	15	7.31	7.35	0.04	63.71	38.00	25.71	6 min.
38	15	7.30	7.43	0.13	39.52	36.69	2.83	6 min.
39	15	7.32	7.40	0.08	47.81	44.23	3.58	6 min.
40	15	7.36	7.51	0.15	52.08	44.54	7.54	6 min.
41	15	7.14	7.33	0.19	47.58	37.48	10.10	6 min.
42	15	7.52	7.52	0.00	54.55	36.88	17.67	6 min.
			Average	.10			10.03	

tunity to breathe through a large unobstructed airway and to overventilate. This period of ventilation was allowed to continue for six minutes in the majority of cases and from ten to twenty minutes in a smaller group, in order to blow off the carbonic acid and allow sufficient time for chemical changes to occur, before collecting the second specimen of blood. After obtaining the second specimen of arterial blood in a like manner, another CO_2 estimate and pH reading with the glass electrode were made to compare with the former tests. We also experimented with a smaller group of animals that were obstructed for fifteen hours. This was done to contrast the chemical effect with that of the twenty-four hour group.

The results of such experiments as these are, like many others, open to technical variations. For instance, fatigue, vitality and exertion exhibited during and immediately before the time of these procedures are varying factors. Dogs which were obstructed for twenty-four hours showed a smaller average decrease of total arterial carbon dioxide than did those which were obstructed for fifteen hours. This difference may have been provoked by a longer period of obstruction, causing a fatigue with inability to manifest as much respiratory activity after removing the obstruction as did the shorter period group. The different periods of time which were permitted the animals for ventilation and resuscitation, prior to obtaining the second blood, did not appear to cause any consistent variations in our findings. The second pH reaction showed an average increase of alkalinity amounting to .13 in proportion to an average decrease of 7.2 volumes per cent of CO_2. There were wider variations between the pH reactions and the CO_2 decrease in some instances, but, in general, the fall of CO_2 consistently favored a greater than normal alkalinity. During the asphyxial stage the arterial blood showed a uniform trend towards acidity but in only one dog did this approach, cross the neutral point of pH 7. There were no clinical indications of respiratory failure in any of the animals after uncorking the airways. Spontaneous breathing occurred and the hyperpnea gradually receded with a trend toward normal rhythm.

DISCUSSION

It is obvious that many factors may be concerned in the production of death associated with obstructive asphyxia. Yandell Henderson[6] has the following to say: "I did not realize as I do now that carbon dioxide deficiency in many cases was due not so much to overbreathing as to the decreased production of carbon dioxide consequent on depressed vitality and lowered muscle tonus." This

point has been well emphasized by Kernan and Barach,[7] in their use of helium in obstructive dyspnea. They have noted that in certain instances, cases approaching exhaustion may suddenly cease breathing when subjected to the most trivial manipulation.

There is no agreement that the abnormal retention of carbon dioxide does act as a respiratory stimulant in the presence of anoxia. Oxygen will restore the carbon dioxide to its normal quantity provided the tissues have not been too severely damaged. According to Schmidt,[8] carbon dioxide is actually a respiratory depressant when the oxygen content of the blood has been lowered sufficiently to cause oxygen crisis; that is, insensibility, apnea and relaxation. Extreme oxygen deficiency depresses respiratory sensitivity and, in more severe instances, produces cerebral edema. In cases known to have died of anoxia, necropsies have shown minute hemorrhages scattered throughout the respiratory center.

SUMMARY

1. The apnea caused by mechanical asphyxia in animals duplicates the human picture of obstructive anoxia.

2. The lowering of arterial carbon dioxide tension suddenly in the animal did not apparently induce any serious or fatal effects upon the respiratory center.

3. In the presence of an acutely precipitated alkalemia and a sudden drop in CO_2 tension, respiration may be disturbed. However, our animal experiments did not indicate that death could be induced by this phenomenon alone.

(We are indebted to Drs. Frank S. Apperly, R. J. Main and H. B. Haag of the Medical College of Virginia for their most helpful and practical suggestions.)

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LXXXII

MULTIPLE PAPILLOMA OF THE LARYNX, TRACHEA AND LEFT BRONCHUS IN A CHILD*

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The extension of papilloma of the larynx into the trachea, especially around the tracheal fistula, when the patient wears a tracheotomy tube, is not uncommon, but extension to the bifurcation and into a bronchus is not so frequently observed.

W. S. Syme[1] reported removal of papillomata from the larynx of a woman, aged 42 years, where there was no recurrence in the larynx, but one year later she returned with a mass of papillomata blocking the right bronchus and partially blocking the left bronchus.

A. M. Zamora[2] reported a case of laryngeal papilloma in a man, 24 years of age, operated by direct laryngoscopy in March, 1928. Seven months later he returned with the larynx clean but with multiple papillomata in the trachea and left bronchus.

Henry Orton[3] reported two cases of papilloma of the bronchus in children, to this Society in 1932, with bibliography appended and remarked "the literature available on papilloma of the bronchus is very meager."

John D. Kernan[4] reported a case of congenital papilloma of the trachea to this Society in 1936.

R. M. Lukens,[5] in the same year, reported a case of multiple papilloma of the trachea in a boy, seven years of age, who had no involvement of the larynx.

The case which I wish to report has a typical history of papilloma of the larynx. The mother noticed for one and a half years that the child was gradually losing his voice and the respirations becoming wheezy, always worse at night, but she did not consult a physician. Finally, when dyspnea became so marked that he was gasping for air, she took him to the McKeesport Hospital where a

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tracheotomy was performed to relieve the obstructive dyspnea and cyanosis.

March 1, 1937, five months after the tracheotomy was performed, the child, a boy, three years of age, was admitted to the Presbyterian Hospital for diagnosis and treatment. Direct laryngoscopy revealed the glottis full of papillomata and no air passing through the larynx. Seven direct laryngoscopies, with removal of the papillomata, restored the lumen of the larynx so that the boy could breathe through his mouth and had a good voice. The aunt of the boy learned how to take care of and change the tracheotomy tube before he was discharged from the hospital April 20, 1937.

This patient returned twice during the summer as an out-patient for observation and direct laryngoscopy. Each time the larynx was clean and the voice good.

October 26, 1937, five months after being discharged from the hospital, he returned so dyspneic and cyanotic that we almost despaired of saving his life. There was scarcely any air through the tracheotomy tube, though the tube was unobstructed by secretion, and introduction of a 4 mm. bronchoscope through the tracheotomy fistula or lower bronchoscopy revealed papillomata filling the lumen of the trachea and left bronchus. These were reamed off the tracheal and bronchial walls with the bronchoscope and removed with the aspirating tube. The introduction of a No. 2 cane-shaped tracheotomy tube was necessary as the curved tracheotomy tube would not reach below the obstruction to relieve dyspnea. Seven additional lower bronchoscopies were required over a period of three months to remove the papillomata from the trachea and left bronchus. Three months after the introduction of the cane-shaped tracheotomy tube, it was replaced by the full-curved tube and there has been no recurrence of papilloma below the larynx.

The interesting part of this stormy period of four months was that the child's life was saved dozens of times by the marvelous care and prompt action of the nurses. The patient was in the children's ward with no special nursing care, and since he was wearing a tracheotomy tube could make no outcry.

Due to the encroachment of the papillomata on the lumen of the trachea and bronchus, a very small amount of secretion would obstruct the breathing and the child, while asleep or at play, would suddenly become cyanotic. This necessitated changing the outer tube (due to the short inner cannula of the cane-shaped tube), using suction, administering oxygen at times, and introduction of a clean

tracheotomy tube. This occurred on an average of seven to ten times every day, and one night the tube was changed twenty times by actual count. This was frequently an emergency, necessitating quick action to save his life.

March 15, 1939, the patient was discharged from the hospital, with no papilloma in the larynx, trachea or bronchus, and a good strong voice. He has had no recurrence of growths in the larynx since December, 1938.

From the history of this case, one would expect to see a puny child with deformed or poorly developed chest but, on the contrary, he is a robust, well developed, healthy boy, with a very cheerful disposition. He still wears a tracheotomy tube, though examination of the larynx May 15, 1939, showed a larynx functioning normally with no signs of recurrence of papilloma and capable of producing a loud clear voice. He will probably be decannulated in the near future.

COMMENT

1. This case corroborates the teaching of Chevalier Jackson that papillomata in children constitute a benign self-limited disease, and if the growths are removed without inflicting any damage upon normal tissue, the child will have a good voice and a normal larynx after the growths have ceased to recur.

2. Although papillomata in the larynx are not uncommon, in the trachea and bronchus they are exceedingly rare. Eternal vigilance and trained tracheotomy nurses are essential to prevent asphyxiation.

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LXXXIII

MYOBLASTOMA OF THE BRONCHUS*

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NEW YORK

In November, 1938, I saw a 15-year-old girl who had been ill for the preceding fourteen weeks. Following the onset of fever associated with cough and purulent expectoration, which were thought to be due to bronchopneumonia of the right lower lobe, she developed clubbing of the fingers and, later, a wheeze in the right lower chest. There were no other signs in the chest. X-ray showed a triangular shadow in the mesial portion of the right lower chest which Dr. Harry Wessler, the consulting thoracic internist who referred the patient to me, interpreted as an atelectatic right lower lobe. At this time the patient began to expectorate blood. Needle aspiration of the right pleural cavity was negative. Sputum examination was negative for tuberculosis. The blood count showed 20,000 W. B. C. and 70 per cent polymorphonuclear leucocytes. In November the temperature still ranged fairly high, 102 to 103 degrees. The cough was very distressing to the patient and it was productive of a great deal of purulent secretion mixed with blood at times.

I bronchoscoped the patient and found the right lower lobe completely occluded by a pinkish-grey, broad-based, pedunculated growth arising from the posterior and mesial aspects of the right lower lobe below the apical branch. After removal of a large portion of the growth by punch forceps, there was a flow of thick non-odorous pus from the distal portion of the bronchus. The diagnosis by Dr Paul Klemperer on microscopy of the excised tissue was myoblastoma.

A second bronchoscopy was done six days days later to remove more of the growth. A small amount of mucoid secretion was found in the right lower lobe bronchus below the lesion. The patient's temperature had dropped to normal and the cough and expectoration were markedly diminished.

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*Read at the 22nd Annual Meeting of the American Bronchoscopic Society, Rye, N. Y., May 26, 1939.

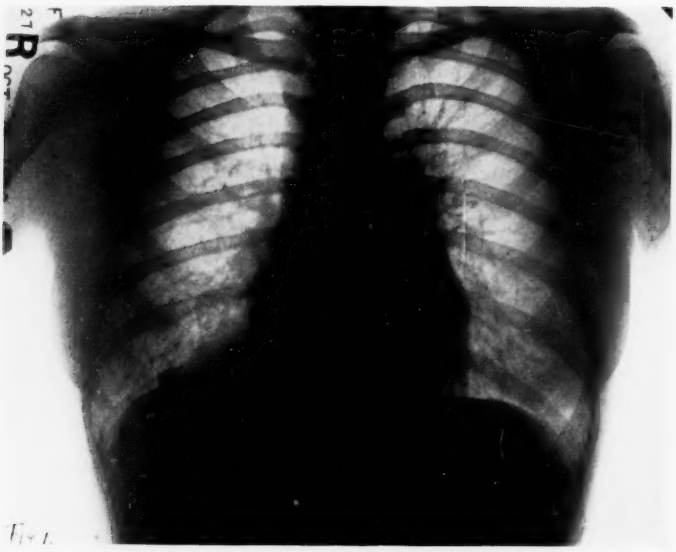

Fig. 1. X-ray before bronchoscopy showing shadow in mesial part lower right chest due to atelectasis.

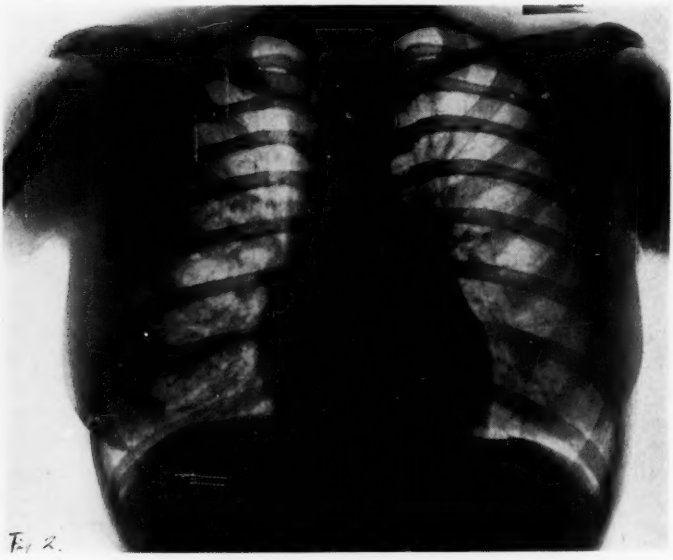

Fig. 2. X-ray four months after onset of treatment showing atelectatic lung expanded.

Six days later a third bronchoscopy was done. The bronchus was clear except for a flat mass on the posterior and mesial aspects of the right lower lobe bronchus. Bipolar diathermic coagulation was applied by means of a fine-pointed active electrode. Three weeks after the first bronchoscopy all symptoms except slight occasional cough had disappeared.

A fourth bronchoscopy, one month after the first bronchoscopy, and nineteen days after the use of diathermy, showed a slight amount of exudate on a depressed area on the mesial wall of the right lower lobe bronchi. Dr. Harry Wessler re-examined the patient on April 26, 1939, five months after the first bronchoscopy. He reported to me that the patient was symptom free, was clinically well, and that on X-ray the right lower lobe had completely re-expanded.

Three features in this history are worthy of emphasis. The first is the presence of lung suppuration, that is, fever, cough, purulent expectoration and signs of infiltration, for fourteen weeks before bronchoscopy was employed in an attempt to discover the underlying cause of the illness. After removal of the obstructing bronchial tumor, the symptoms disappeared fairly rapidly.

The atelectasis persisted for several weeks after the disappearance of clinical symptoms. Five months later the lung was clear on physical and radiologic examination (Figs. 1 and 2). The length of the illness could have been markedly shortened by observation of the dictum that bronchoscopy should be performed as soon as the diagnosis of lung suppuration is suspected. Atelectasis that has persisted as long as it did in this patient does not always clear up. Often it continues and is complicated by bronchiectasis and its dire sequelae.

The second feature to be emphasized is the nature of the tumor, myoblastoma. This is the first reported case of this tumor occurring in the bronchi. Myoblastoma was first described as a definite entity in 1926 by Abrikosoff. Since then somewhat less than one hundred cases have been reported. They have been found most frequently in the tongue, jaw, larynx, esophagus, lacrimal apparatus, skin and breast. They are composed of large polygonal cells and ribbon-like syncytial masses. The cytoplasm is coarsely granular and basophilic. The nuclei are either rounded or elongated and may be situated centrally or peripherally. The granules may be closely packed or loosely arranged so that they resemble xanthoma cells (Fig. 3). The use of fat stains will settle the question of this diagnosis. There may be ribbon-like syncytial structures of varying lengths with the coarse basophilic granules and with several nuclei. The granules are usu-

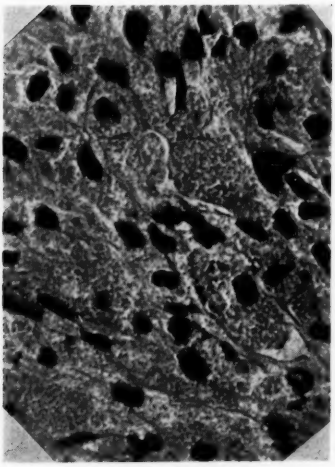

Fig. 3. Higher magnification showing the large polygonal tumor cells with strikingly granular cytoplasm.

ally irregularly arranged, but occasionally they are placed in parallel rows which suggest the cross striations of voluntary muscles. They are generally benign tumors, although a few instances of malignancy, either local or metastatic, have been reported. These tumors are thought to originate from primordial voluntary muscle cells and their presence in close association with striated muscles is readily understandable. In order to explain the presence of myoblastoma in regions where striated muscle does not occur normally, a dysontogenetic origin of the cells is postulated. This implies the dragging along of the primordial cells by the other mesenchymal cells when the latter migrate to the organ to which they belong. Thus the presence of primordial striated muscle cells in the bronchi can be explained by the inclusion of these cells in the descent of the bronchi from the digestive tract which has striated muscle in its walls.

The treatment employed was removal of the growth by punch forceps and subsequent diathermic coagulation of the base of the tumor. The last bronchoscopy showed the bronchial lumen to be patent and no evidences of tumor or disease to be present.

121 East 60th St.

LXXXIV

THE ACUTE CHEST FROM THE BRONCHOSCOPIC STANDPOINT

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It is our privilege in the practice of bronchology, as Chevalier Jackson has so aptly named this specialty, to see the acute chest in the very early stages and to encounter symptoms and physical findings which are rarely seen or recognized by medical men outside of this specialized field. Because of this privilege, it behooves us to work in close co-operation with these other medical men with the hope that better service for more patients, especially in private practice outside medical centers, may result.

The findings as we see them in the early ball-valve type of obstructive emphysema, secondary to foreign bodies, are contradictory to what is seen by general physicians in their everyday experience with respiratory infections. In such instances we may examine patients, which, at the time, have few respiratory symptoms, but breath sounds may be deficient or absent over a part or whole of a lung. This same side of the chest may be tympanitic to percussion and be clearer than the other side in the x-ray film. Such findings just do not make sense in the minds of practitioners who associate deficient or absent breath sounds with dullness to percussion and positive x-ray films. Naturally in these early cases of partial foreign body obstruction of a bronchus, the casual roentgenologist may report positive findings on the uninvolved side of the chest because by contrast the emphysematous lung is clearer in the film. It is natural that such a report from x-ray studies further tends to confuse the possible diagnosis in the minds of the medical men whose experiences with foreign body obstructions are rare. It is also natural that many infants and young children are given expectant medical treatment in this early stage until in a few days, the symptoms, physical signs and x-ray evidence "fit the picture" of pneumonia. By this time there is often no confusion in the minds of the casual roentgenologist and many general physicians. Pneumonia it is and the recognized expectant treatment is prescribed. Often everyone is satisfied but the mother of the patient, who still keeps insisting

that her child was perfectly well until it suddenly choked while eating peanuts or popcorn. In some instances, parents have been rebuked by their medical attendants for talking about the possibility of a foreign body because the x-ray film did not show any, but did show pneumonia. At times parents have been advised that if pieces of peanut were present in the air passages, they would be absorbed.

Diagnostic misunderstandings are not confined, as we all know, to this early acute chest which is secondary to foreign body obstruction, but continue to be a problem for a few days, weeks or months after onset of symptoms and a pathological picture which often represents pneumonia to the general physician and obstructive atelectasis to the bronchoscopist. Unless the physician who is treating children with acute chest conditions, can visualize the important part played by mechanical obstruction in the air passages, whether from a real foreign body or plugs of mucus, it is instinctive as a result of previous training and experience, to consider the symptoms and physical findings as evidence only of infection of the bronchial and lung tissues.

Broncho-pneumonia has appeared in our literature very often and for a long time and has naturally been accepted by most physicians as a scientific diagnosis of a certain symptom complex. It is reasonable to assume that in most instances this diagnostic term suggests to the medical mind thoughts of the type and virulence of the infective organism and the possible specific treatment which may be directed toward this particular organism. Surely there are some children with acute chest conditions in whom the diagnosis of the probable cause of their disability is determined by chance; broncho-pneumonia, if under the care of a general physician; atelectasis if in the service of one doing bronchoscopy. I hasten to add before I am considered radical, that of course there are many, many cases in which the diagnosis should be broncho-pneumonia and the patient should be treated medically, but I also insist that much so-called broncho-pneumonia is really atelectasis in which the mechanical obstruction of a bronchus is of much more importance than the infective process.

It also seems reasonable to some of us who are doing bronchoscopy, to consider many of the chronic conditions, especially bronchiectasis in older children and young adults, as natural end results of too much emphasis having been placed upon infection and medical treatment and too little emphasis upon mechanical obstruction and its removal, during the stage when the acute condition was being treated.

In passing, and in the light of fairly recent experiments and reports concerning chest findings after the use of various oils, silver preparations, etc., in the nasal passages, it does seem possible that some of the cases of so-called broncho-pneumonia in the young child may result because of the too active treatment of the noses of these children in the early stages of what is commonly a descending infective process. We all know that most of the children with upper respiratory infections are seen in the homes by general physicians and pediatricians and naturally, most of the nasal drops are prescribed by them and not by otolaryngologists. Most of these drops are used by the mothers with little chance for control of the position of the little patients. I think that most of us in this specialty feel that very little good and possibly some harm results from the habitual use of various medicinal agents in the noses of infants and young children.

The child who is destined to develop a serious descending infection of the respiratory tract is probably fortunate if symptoms of laryngeal obstruction are of sufficient importance early to direct attention to the possibility of mechanical obstruction. Laryngeal obstruction is a dramatic episode and suggests a possible emergency to any medical man in charge of the case. Usually descending tracheo-bronchitis without serious laryngeal symptoms is considered a medical problem and is often treated by expectant methods. In my opinion, most of the respiratory infections in children are caused by ordinary bacteria of ordinary virulence. Severity of the infective process is commonly of much less importance than the combination of mechanical obstruction and lack of cough reflex. We in this specialty who have experience with foreign bodies in the respiratory tract, should use this experience to help the other medical men interpret similar findings which may follow the formation of masses or plugs of mucus in the course of ordinary descending respiratory infections.

I am calling attention to some of these misunderstandings concerning the diagnosis and treatment of the acute chest, especially in infants and young children before this special group, not because anything I say is new to you, but because I feel that we might possibly stand medically indicted for not having worked harder, longer and more collectively, to broadcast in general medical meetings and the general literature, what we have learned because of our experience in bronchoscopy. Naturally, this chance to visualize the respiratory tract from within is being denied to most general physicians and pediatricians.

It is my opinion that too much of the literary output from this and other special societies is prepared for consumption and understanding by a limited number of the members of our profession who are working in a special field. We continually coach each other concerning rare and unusual conditions in our particular specialties, but too often expect the general medical man to know about the possibilities of diagnosis and treatment which are outside his everyday experience, although they are classed as routine procedures in large clinics and medical centers.

Case reports, statistics and prints of x-ray films are presented with the hope that some helpful hints in relation to differential diagnosis and treatment of certain acute chest conditions may be gleaned, especially by medical men outside our specialty. These physicians are naturally seeing, in their everyday practice, most of the patients whose chest conditions are under consideration in this paper.

Massive Atelectasis: Collapse of lung and chest, postoperative. The patient, a male, age 19, was operated upon under ether anesthesia, for repair of an inguinal hernia. That evening there was complaint of pain in the left chest and fever of 101.6 degrees. By the next day there was cough, pain and tightness in the chest, fever of 103.8 degrees and leucocytosis of 20,600. These symptoms persisted, with increase of dyspnea. Roentgenogram taken on the seventh postoperative day, showed massive atelectasis, left chest. There was dullness over the entire left chest with almost complete absence of breath sounds on this side. The pulse was rapid and the patient looked pale and distressed and was evidently acutely ill.

Bronchoscopy was done under local anesthesia on the eighth postoperative day. The left main bronchus was found collapsed, with mucosa thickened and dusky red. There was a small amount of exudate but no real plug. The bronchus opened before the bronchoscope like the esophagus. Mucus was removed by suction.

After bronchoscopy, thick mucus was coughed out. Patient immediately felt better and had less dyspnea. Breath sounds were plainly audible over the left chest. All symptoms cleared rapidly and the patient was dismissed four days after bronchoscopy. Chest picture was taken on third day after bronchoscopy and shows aeration of left lung, with shift of heart back to the right.

Comment: Here was a typical picture of massive atelectasis of the chest and lung after hernia operation under ether anesthesia. No plug was found in the bronchus as was to be expected and is usually found in such cases, but rather a collapse of the main bronchus

itself. The cough and muscular exertion incident to bronchoscopy under local anesthesia probably played the greatest part in re-establishing the function of the left lung. Early in the week before bronchoscopy, change of posture in bed or some manual treatment such as a blow on the back with the fist, probably would have relieved the symptoms, as so commonly happens in cases with this post-operative complication. In this instance, bronchoscopy rapidly changed the clinical course when expectant medical treatment had failed. Massive atelectasis after surgical procedures, especially under general anesthesia, is not a rare condition. It represents the modern counterpart of what was called "ether pneumonia" for so long. Good results are obtained very commonly, when manipulation from without or bronchoscopy from within are used, in an effort to relieve mechanical obstruction before secondary infection develops.

Lung Collapse: This roentgenogram shows collapse of the lung itself after traumatic pneumothorax from fracture of ribs. The patient was struck by an automobile and was admitted to the hospital in shock. Air was removed by needle several times with rapid recovery clinically and with reference to chest findings.

This roentgenogram is included to contrast the appearance of pure lung collapse with collapse of lung and chest which is present in massive atelectasis. Treatment naturally was entirely different and is entirely foreign to the basis of the discussion in this paper. It does, I think, help us to visualize the difference between two chest conditions which may easily be confused in our minds if physics is not considered in relation to pathology. In massive atelectasis there is collapse of the lung and chest with heart and mediastinum drawn to the affected side, diaphragm elevated and rib interspaces narrowed.

Foreign Body Chest (Early). Ball-valve emphysema. Child, 1 year of age, choked while eating peanuts 24 hours before admission to the hospital. There was cough, some cyanosis and wheezing respiration.

There was abdominal breathing and poor expansion of the right chest. Breath sounds were practically absent over right lung, emphysematous over the left one. Percussion tympanitic over both lungs.

X-ray of chest (Fig. 1). Right lung emphysematous, heart shifted to left, diaphragm depressed on right on both inspiration and expiration.

These findings suggested the presence of a foreign body in the right bronchus which allowed some air to enter the lung on inspira-

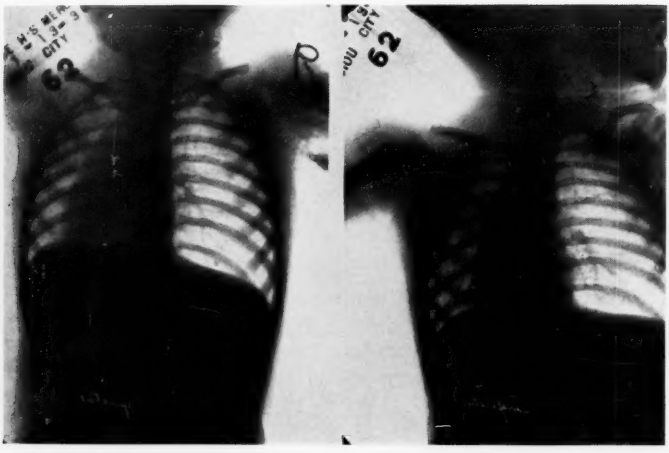

Fig. 1. Ball-valve emphysema, right lung. Heart shifted to left, right diaphragm depressed on inspiration and expiration. (Peanut in right bronchus.

tion but prevented it from getting out during expiration, so-called ball-valve emphysema.

Bronchoscopy: No anesthetic. Portion of a peanut was removed from the right bronchus along with some exudate by suction.

Roentgenogram taken 24 hours after bronchoscopy showed normal relations of heart, lungs and diaphragm. Recovery was rapid and complete.

Comment: Such association of symptoms, physical signs in the chest and x-ray findings, can only be produced by a sudden interference with function such as comes after inhalation of a foreign body. The regular progression of events in a descending process due to infection is well understood. If these signs of early chest involvement were more clearly understood and recognized by general physicians, pediatricians and roentgenologists, it seems likely that there would be fewer late involvements such as atelectasis or lung abscess and there would be fewer foreign body cases treated as pneumonia.

Foreign Body Chest (Early): Massive atelectasis. Represents a massive atelectasis which developed early after a foreign body (pea-

nut with mucus) apparently blocked the bronchus completely. The trapped air was absorbed and rapidly replaced by fluid.

This patient, a child 2½ years of age, was brought to the hospital in a moribund condition, respiration had ceased. The x-ray had been taken two days previously and the patient was being treated for pneumonia. A bronchoscope was passed, some pieces of peanut and thick sticky mucus were removed from the left bronchus, but child could not be revived by artificial respiration.

Massive atelectasis after inhalation of a foreign body is comparatively rare. Usually some air enters the lung or part of it, past the foreign body, or the foreign body changes location in the trachea or bronchi from time to time. In this instance the child was given expectant medical treatment for pneumonia until it was too late to do anything else.

Foreign Body Chest (Late): Atelectasis. The Patient, a child 6 years of age, came to the hospital with the history that nine weeks previously she had choked while eating cracker-jack. There had been no respiratory symptoms previous to the choking spell. There was some cough and wheezing immediately. In a few days, fever accompanied the respiratory symptoms and a diagnosis of pneumonia was made by the attending physician. Cough and fever continued for weeks with emaciation, food refusal and night sweats.

The patient was examined by a surgeon three weeks after the sudden onset of symptoms. He made a diagnosis of empyema and resected a rib. No pus was found.

Consultation was held by three physicians five weeks after the acute onset. They decided they were dealing with tuberculosis, though no organisms could be found.

The patient was brought in for bronchoscopic examination by the parents because they had felt all along that the trouble had started when the child choked while eating cracker-jack. The attending physicians discouraged their coming and informed them that the x-ray showed no foreign body. The parents were also informed that if a peanut had been present in the lung, it would have been absorbed.

Examination (nine weeks after choking spell) disclosed an emaciated child with a flat left chest and very poor expansion on this side. There was cough with some sputum. The temperature was 101 and the white count 22,000. There was dullness over the left

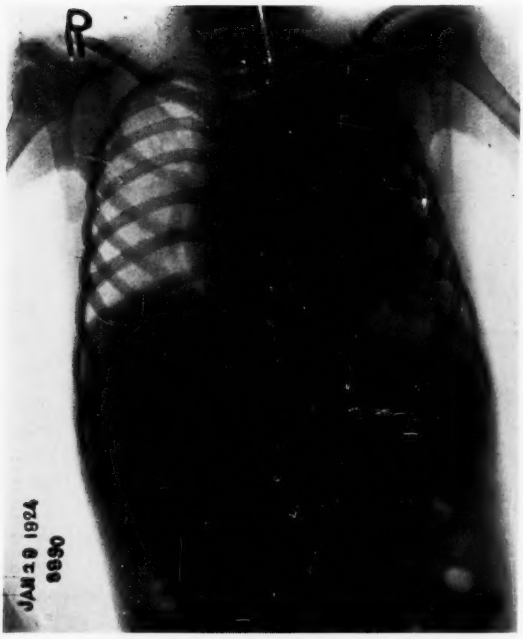

Fig. 2. Atelectasis or drowned lung, left.
(Peanut in bronchus nine weeks.)

chest except over apex. Breath sounds were very clear in the right chest.

X-ray of chest (Fig. 2) shows atelectasis (drowned lung) left with a small clear area at the apex.

Bronchoscopy: no anesthetic. Pieces of peanut held together by sticky mucus were found in the left main bronchus. When this mass was removed, there was a gush of purulent material through the bronchoscope. More material was removed by suction. There was rapid improvement in the general condition and physical findings in the left chest.

A second bronchoscopy was done after four days. Some mucus was aspirated. Breath sounds appeared in the entire lung, expansion of the chest increased and general condition improved rapidly. Pa-

tient was dismissed on the twelfth day and made a rapid and complete recovery with no sequelae.

Comment: A well child choked while eating cracker-jack. She was treated for pneumonia; operated for possible empyema; diagnosed as tuberculosis; and recovered after removal of pieces of peanut and mucus by bronchoscopy nine weeks after the acute onset.

Foreign Body Chest: Upper lobe atelectasis. Child, 1½ years of age, choked while eating candy 48 hours before admission to the hospital. Noisy respiration but no cyanosis. During recent hours there was fever, rapid respiration and food refusal.

Examination of the chest showed dullness to percussion and diminished breath sounds over the upper right chest with a few rales: good breath sounds and tympanitic percussion right lower and all of left chest. Emphysematous breathing over entire left chest.

W.B.C. 22,000. Temperature 101 degrees.

X-ray (Fig. 3, View 2): Atelectatic area right upper chest. Lower right lung and entire left lung clear.

Naturally, the above physical findings were rather typical of lobar pneumonia, but the history suggested an acute onset in a previously well child after a choking spell while it had candy in its mouth.

Bronchoscopy (no anesthetic), almost half of a small peanut was removed from the upper portion of the right main bronchus.

X-ray (Fig. 3, View 1) two days after bronchoscopy shows clearing of atelectatic area.

Comment: The rare finding of upper lobe atelectasis and clear lower lung with a vegetable foreign body in the main bronchus was apparently explained as follows: The curved portion of the half peanut was forced against the opening of the upper lobe bronchus while the flat side allowed air to pass between it and the wall of the main bronchus into the lower lobe. Recovery was rapid and complete. Physical findings and x-ray evidence would easily have suggested lobar pneumonia unless close attention was given to the history of foreign body inhalation.

A child, 10 years of age. Tonsillectomy under ether anesthesia had been performed by a general physician six weeks previous to examination. There had been some cough and fever with loss of

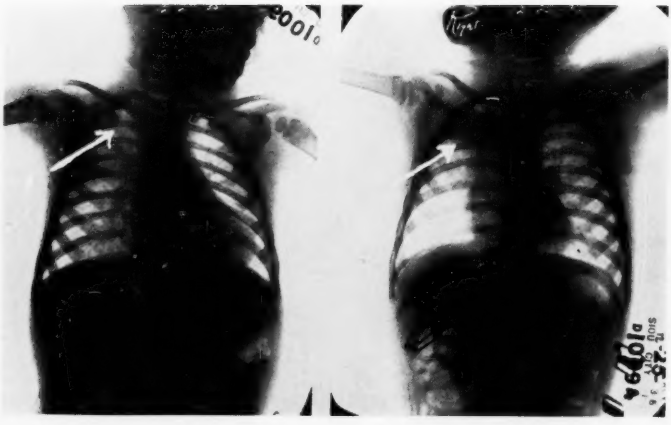

Fig. 3, View 2. Same chest two days after bronchoscopy and removal of peanut. View 2 is on left side of print.

Fig. 3, View 1. Upper lobe atelectasis, right. (Peanut in right bronchus.) View 1 is on right side of print.

weight since the operation. Patient had not improved after rest in bed.

X-ray (Fig. 4, View 1) showed a clearly defined upper lobe atelectasis on the right. The group of opacities near the hilus were reported as calcified areas, probably in glands. No opaque foreign body was reported. The diagnosis was lung abscess. The pediatrician in charge of the case asked for bronchoscopy.

Bronchoscopy: There was no obstruction in the right main bronchus nor was there any inflammation of the mucosa or any mucus present. The opening of the upper lobe bronchus could be seen clearly and no inflammation or discharge were evident. A flexible suction tube was passed into the bronchus but no secretion was obtained and no obstruction was encountered.

The child was kept in bed at home for two weeks. There were very few symptoms and appetite was good. There was slight fever in the afternoons and some irritative cough.

The patient returned to the hospital for study. That evening there was a spell of coughing and a small tooth was expectorated.

X-ray (Fig. 4, View 2) shows now one less opacity in the group at the hilus which were believed to represent calcified areas in glands. Recovery has been fairly rapid and apparently complete

Comment: The record of this patient is presented for these reasons:

1. Symptoms were quite typical of lung abscess following tonsillectomy.

2. Location of the lung involvement in a portion of the upper lobe was rare under the circumstances.

3. The presence of an opaque foreign body was not recognized in the x-ray films by ourselves, two roentgenologists, a pediatrician or the family physician.

4. Spontaneous expulsion of the tooth relieved the patient of her disability and the attending physicians of the responsibility of subsequent treatment. Even with the proper diagnosis the mechanical problem of removing this smooth tooth from the upper lobe bronchus would not have been easily solved. The happy ending in this particular instance cannot, we hasten to add, give any assurance to those who might want to consider expectant treatment as a possibility in the handling of cases with foreign bodies in the air passages. Nature is not always kind and not necessarily tolerant of our human failings.

Because we have heard and read so much about the severe reaction of the mucosa of the respiratory tract to the presence of the peanut, it seems there should be some interest in the presentation of patients occasionally, in which this expected reaction did not occur and in which x-ray examination of chest was negative.

Foreign Body Chest: Negative X-ray Findings. Peanut in bronchus 3½ days. Patient, age 22 months, three days before examination, choked while eating peanuts. There was some cough and moderate dyspnea; most noticeable finding was a wheezing respiration.

Examination: Wheeze at mouth, breath sounds diminished right base; no rales; no dullness; normal expansion; temperature 98 degrees, W.B.C. 9,700.

X-ray of chest: Negative.

Bronchoscopy: No anesthetic. Large piece of peanut, practically obstructing the right bronchus, was removed. There was very slight reaction in the mucosa and very little mucus.

Recovery rapid, dismissed on third day. No sequelae.

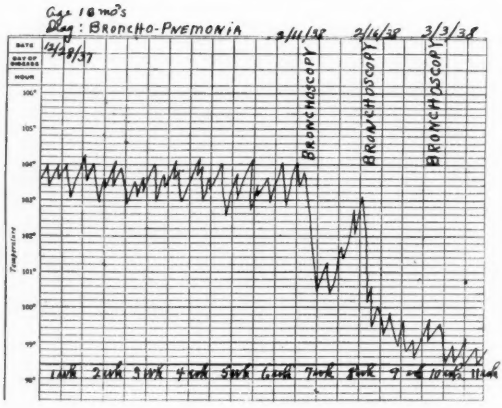

Fig. 4. Chart of "broncho-pneumonia" treated by bronchoscopic aspiration.

Foreign Body Chest: Peanut in respiratory tract nine weeks. Patient, age 22 months, was perfectly well until there was choking while eating peanuts. For a few days there was a peculiar wheezing respiration with cough, then fever and malaise.

Family physician made a diagnosis of pneumonia and started expectant medical treatment. (During all this time the mother insisted that the trouble started when the child choked while eating peanuts.)

Again there was gradual improvement, but there was some loss of weight and cough.

Six weeks had gone by since the original choking spell and again acute symptoms of cough, fever and food refusal developed.

Family physician again made a diagnosis of pneumonia and started expectant medical treatment. (Mother again insisted that the troubles all started when the child choked while eating peanuts, but was informed by the attending physician that the x-ray did not show any foreign body and if it had been a peanut, it would have absorbed.)

During the ninth week of the disability the patient came for possible bronchoscopic study, because the mother of the patient

whose chest plate appears earlier in this paper, as (Fig. 2) lived in the same town and had heard the history of this particular case.

Examination: Child of 22 months, in surprisingly good physical condition considering the history of three attacks of pneumonia (so-called) within nine weeks. There was some emaciation, pallor and moderate cough. Breath sounds were fairly clear over the entire chest with a few moist rales. Expansion good, no dullness. Temperature 100 degrees. W.B.C. 10,000.

X-ray of chest: Negative.

Bronchoscopy: No anesthesia. A large piece of peanut was found in the right bronchus and removed. There was practically no swelling or inflammation of the mucosa of the trachea or either bronchus. The piece of peanut did not crush in the forceps and resembled a piece of raw potato.

Recovery was rapid and complete.

Comment: The nine week sojourn of this piece of peanut in the respiratory tract of an infant of 22 months without more serious consequences is probably explained by the fact that the smooth, raw, half-peanut remained intact and shifted from one or other bronchus to the trachea between the attacks of so-called pneumonia. The reaction in the mucosa of the air passages was slight with only a moderate amount of mucus.

X-rays of chest: Negative.

Broncho-Pneumonia: Report of case treated by bronchoscopy. The chart (Fig. 4) represents the clinical course of an infant of 10 months. Broncho-pneumonia is of course a real clinical entity, but to some of the members of our profession who happen to look from within the respiratory tract, the so-called pneumonia is often atelectasis secondary to mechanical obstruction of a bronchus by thick mucus or plugs of sticky or inspissated material. The symptoms and physical signs are often, we believe, more dependent upon the obstructive or mechanical element than upon the severity of the infection or the type or virulence of the organisms present. Naturally, the bad effects of mechanical obstruction are more evident in the infant because the protective cough reflex which is so active in older children and adults is often absent or feeble in the baby or young child.

After an ordinary respiratory difficulty the child ran a course as charted, of high continuous fever, leucocytosis, food refusal and

emaciation for over six weeks. During this time the diagnosis was broncho-pneumonia and the treatment was medical and expectant.

X-ray report: Consolidation of lower lobes, more complete on the left. The roentgenologist advised bronchoscopic examination in the seventh week of the broncho-pneumonia.

Bronchoscopy: No anesthesia. The trachea was negative. There was some mucus in the right bronchus. In the left main bronchus there was a plug of glistening white material which seemed to be filling the lumen. This plug was removed with a forceps and thick muco-pus was removed by suction.

Clinical course: There was a drop in temperature and less dyspnea for three or four days, then recurrence of the previous symptoms.

Bronchoscopy (second) five days after first. Again a similar plug of sticky white material was removed with a forceps from the left bronchus and thick muco-pus by suction.

As the chart indicates, there was a rapid drop in fever and there was also less cough, restlessness, food refusal and leucocytosis. The baby began to gain weight rapidly and appetite increased.

Bronchoscopy (third) about two weeks following the second. At this time there was no plug but a small amount of mucus was removed by suction.

The recovery was rapid and complete with no sequelae.

Comment: Broncho-pneumonia which runs an extended course is probably often really atelectasis secondary to mechanical obstruction of a bronchus. In such instances the mechanical obstruction is of probably much more importance than the infective process. Treatment in many instances of delayed resolution should be active by bronchoscopy and not always passive or expectant as it naturally should be in many early infective conditions.

Laryngo-Tracheo-Bronchitis: Traumatic. During a period of seventeen months, eight consecutive patients from one to two years of age came under our care because of a possible foreign body in the air passages but no foreign body was found. During this period not one foreign body was removed from the trachea or bronchi but the usual number were encountered in the larynx and esophagus.

In each instance there was a history of a sudden onset of choking, followed by varying degrees of cyanosis, stridor, cough, etc., in a child with no previous respiratory symptoms.

Popcorn, peanuts, coffee beans, field corn and watermelon seeds were the materials found in the mouths of these children when the acute respiratory symptoms developed. In all cases there was some degree of dyspnea with supra-sternal retraction, wheezing respiration and cough; in some there was cyanosis.

Deficient breath sounds over a tympanitic lung suggested ball-valve obstructive emphysema in several chests; deficient breath sounds over areas of dullness suggested atelectasis in others.

Temperature ranged from normal to 104 degrees; W.B.C. from below normal to 39,000.

X-ray findings: In two instances films were not taken or were not clear. The report was negative in one instance. There was evidence of ball-valve obstructive emphysema in two chests; and pneumonitis or consolidation in three.

The patients in this series came under observation in from two hours to ten days after acute symptoms developed.

Bronchoscopy: in each instance and without anesthesia. No foreign body was found in any of the cases. It is very likely that pieces of material too small to be identified, were present in the mucus or had been drawn deeply into the smaller bronchial divisions. Mucus, sometimes in sticky masses or plugs, was removed by suction in each case from one or both bronchi and the trachea. In one child of two years, coming under observation ten days after the original choking spell, there was a plug of mucus practically obstructing the bronchus. In this case a second bronchoscopy with removal of mucus was done eight days after the first one. Each patient improved after bronchoscopy; none were made worse. Tracheotomy was not necessary in any instance. All patients were kept in croup tents was high fluid intake and routine general care.

Result: Recovery in all eight patients without sequelae. Six patients were dismissed within four days following admission; one was under observation eight days; and one twelve days. In this latter case, recovery was slow and a second bronchoscopy was done before dismissal. These patients have all been followed from three to eighteen months after bronchoscopy. In several instances roentgenographic evidence of pneumonitis persisted long after the clinical symptoms had disappeared. No sequelae are now present in any of the eight patients.

Comment: Eight consecutive cases were encountered with typical symptoms of a foreign body in the air passages and typical physi-

cal signs, but no foreign body was found at bronchoscopy. No respiratory symptoms were present prior to the sudden choking spells.

Mucus, thin, bloody or in sticky masses or plugs was removed by suction through the bronchoscope in each instance. A second bronchoscopy was necessary in one case; tracheotomy was not necessary in any instance.

Recovery was prompt in six cases, delayed in two, complete in all without sequelae.

Does the experience with this group of consecutive cases represent pure coincidence of rare associations as is often encountered in medical practice?

May we assume that general physicians are more alive to the importance of the possibility of a foreign body in the air passages and refer more patients earlier, even when it might seem from the symptoms and physical signs that the foreign material had been expelled by coughing or vomiting?

Do the findings and good results in this small group of similar rare cases suggest possibilities in the treatment of some patients with tracheo-bronchitis or broncho-pneumonia (atelectasis) by bronchoscopic aspiration, even though there is no definite history of foreign body inhalation?

Does this experience warrant the assumption that traumatic laryngo-tracheo-bronchitis, secondary to the temporary sojourn of a foreign body and the reactions of the tissues to this substance, or secondary to the presence of pieces of foreign material too small to identify, is a clinical entity of some importance?

Naturally the possibility that the foreign body was overlooked in some of these cases will come to the mind of the reader. The only answer is that each and every patient recovered without sequelae, which is all one can expect whether or not a foreign body is actually identified and removed.

Laryngo-Tracheo-Bronchitis: Traumatic and Infective. Child 2 years of age choked with field corn kernels in mouth eight hours before admission. There was a croupy cough, hoarse cry, wheezing respiration and some cyanosis present. No history of previous illness.

X-ray showed good respiratory excursion, hypo-ventilation of the left lung, indicating slight obstruction of the left bronchus.

Bronchoscopy: Several particles of a kernel of corn were removed from the lower portion of the trachea. Bronchi clear after removal of mucus by suction. Mucosa of trachea and bronchi was congested with some edema.

Dyspnea became more pronounced 16 hours after bronchoscopy and tracheotomy was done. Bronchoscopy through the tracheotomy wound disclosed no foreign body, but some mucus was removed by suction.

Routine instillation of normal saline into the tracheal cannula followed by suction with the catheter was carried out day and night. Fluids were forced and the patient was kept in a croup tent.

There was a gradual improvement in all symptoms and the canula was left out during the daytime by the tenth day.

At this time the father and mother who had alternated in staying with the child became ill, the father with acute follicular tonsillitis and the mother with acute pharyngitis. Every effort was made to isolate the baby from the parents. Two days later the child's temperature reached 104 degrees (from normal) and dyspnea returned.

The tracheal canula was replaced and kept in constantly. Routine suction, forced fluids, etc., were again used as in the early days following bronchoscopy. Temperature and leucocytosis remained high and general condition became worse.

X-ray: Extensive broncho-pneumonia, bilateral.

Death five weeks following bronchoscopy for removal of foreign body.

Comment: The infant developed symptoms of a foreign body in the air passages following a sudden choking spell with pieces of corn kernel in mouth. A bronchoscopy was performed with removal of the foreign bodies.

Tracheotomy because of continued dyspnea. Result good for ten days after use of routine suction and supportive measures.

Father and mother developed acute throat infections and insisted on close contact with the child.

Patient developed high fever, dyspnea, new evidence of infective tracheo-bronchitis. Tracheal canula had to be replaced and routine suction continued. Broncho-pneumonia and death.

Traumatic tracheo-bronchitis (severe type) secondary to the sojourn of a foreign body was controlled by tracheotomy and routine suction for ten days; infective tracheo-bronchitis apparently secondary to contact with a virulent organism harbored by the parents caused broncho-pneumonia and death despite all efforts to keep the lower air passages clear by suction through the tracheal canula and by bronchoscopy.

Laryngo-Tracheo-Bronchitis: Acute, infective. This disease, because of its high mortality rate, deserves considerable respect and attention by everyone in the medical profession, regardless of specialization or limitation of practice. It appears endemically or in epidemics in all parts of the country during any season of the year.

In 1920 we realized for the first time that there were non-diphtheritic infections of the larynx, trachea and bronchi in which the onset and symptoms simulated diphtheria but in which the causative organisms were the streptococcus, staphylococcus or pneumococcus. Diphtheria antitoxin failed to give results. Mechanical obstruction of the air passages below the tracheal canula was much more troublesome than in diphtheria and apparently accounted in the main for the lung involvement and high mortality rate which accompanied this disease.

Forty cases of severe type are considered in this report. They have appeared sporadically in private practice since 1920; never in epidemic proportions.

Tracheotomy was performed in twenty-five of the cases; intubation in none. Tracheotomy has been the chosen procedure, mainly because the canula stays in place and allows of suction to help control the descending mechanical obstructive involvement of the air passages which is the most serious feature of the disease. Liquefying solutions such as normal saline or weak adrenaline are dropped into the canula preceding suction. Suction is repeated every 20 minutes, day and night, regardless of symptoms of dyspnea.

Bronchoscopy was done in many instances, before and after tracheotomy, to investigate for the possible cause of the dyspnea and to remove plugs and crusts which commonly form low down in the respiratory tract.

Mechanical obstruction, due to descending involvement, has been of more importance in producing the high mortality rate than the severity of the infection.

Pathology: Thickened mucosa with a thin exudate, with rarely a real membrane as is seen in diphtheria. Thick discharge is found in the trachea by bronchoscopy or after tracheotomy. Plugs of semi-solid, sticky material are often found in main bronchi or smaller divisions. Atelectasis and broncho-pneumonia are the terminal processes in descending involvement.

Bacteriology: The streptococcus has been predominant with the staphylococcus next in importance. Diphtheria bacilli have not been found except in a few instances where the presence of a few was probably incidental.

Most of the patients have been infants or young children and the severity of the disease and high mortality rate have seemed to depend more upon the lack of cough reflex in the small, obstructed air passages of the baby or young child, than upon the type or virulence of the organisms found.

Two adults have come under observation with this disease; one recovered after treatment in an oxygen tent; the other died after tracheotomy from secondary cardiac exhaustion.

Diphtheria antitoxin has been given to each patient in order to play safe while waiting for a culture report and in the hope that a reaction to the foreign protein might be beneficial. Other foreign proteins or vaccines have not been used routinely.

Postural drainage has been used to some extent in all patients, but a real systematic procedure, as has been reported by others, has not been used. The children are all turned on one side, then the other, during the suction treatments and are not allowed to remain in one position for any length of time.

Sulfanilamide has been available and used in the last eight cases in this series. In this small group, our mortality rate has been just as high and the course of the disease did not seem to be modified. In some instances the mechanical obstruction, as is always likely in this disease, was too serious and acute to allow much time or opportunity for any drug to show to advantage. It is to be hoped that a decrease in mortality rate will follow when this drug is used early and in proper dosage, as determined mainly by the type of causative organism.

Mortality rate in the entire group of forty cases has been 40 per cent; in the tracheotomized cases, 60 per cent. Again I repeat, this disease deserves considerable respect and attention by all members of our profession, especially those who are seeing and caring for

infants and small children. Delay in recognizing symptoms of mechanical obstruction, especially of the lower air passages in these little patients, is a serious error of omission. Watchful waiting and so-called expectant medical treatment often allow the development of atelectasis, pneumonia, cardiac exhaustion and death, when active measures to relieve mechanical obstruction, if begun early and used with persistence, might have prevented a fatal outcome.

SUMMARY

1. The medical man who is privileged, because of his experience in bronchoscopy, to visualize the respiratory tract from within, should be able to obtain evidence and give treatments which can be helpful to the general physician, internist, pediatrican and chest surgeon in the handling of acute chest conditions.

2. The early acute chest secondary to foreign body inhalation gives symptoms, physical findings and x-ray evidence which is entirely different from that produced by the ordinary descending respiratory infections. Sudden trauma to healthy tissues is entirely different from the gradual insult of an infective process.

3. Broncho-pneumonia is probably often atelectasis secondary to mechanical obstruction in a bronchus by masses or plugs of mucus, especially in the infant in which the cough reflex is commonly inhibited or absent. In such instances the infection and the type and virulence of the organisms often are of much less importance than the mechanical obstruction. Bronchoscopic aspiration is available and surely has a definite place in the diagnosis and treatment of some of the conditions which are now called broncho-pneumonia.

4. Bronchiectasis in the older child or young adult is probably often a direct result of insufficient drainage by mechanical means during some acute chest syndromes, when atelectasis was present but infection probably was not of importance.

5. History is of great importance in the proper handling of acute chest conditions, especially in children.

Sudden onset of respiratory symptoms in a previously well child should create suspicions of a possible foreign body.

Many so-called pneumonias and lung abscesses might be prevented if proper attention was given to the possibility of a foreign body as the probable reason for the sudden onset of respiratory symptoms.

6. Laryngo-tracheo-bronchitis (traumatic) is suggested as a clinical entity. Eight consecutive patients with a typical foreign body onset were treated. No foreign body was found in any instance, but after bronchoscopy and removal of mucus by suction, the symptoms, physical findings and x-ray evidence of acute respiratory involvement rapidly disappeared.

It is suggested that probably some patients with bronchitis or broncho-pneumonia (so-called) might also be benefited by bronchoscopic aspiration of mucus or infected material before atelectasis becomes real pneumonia.

7. Laryngo-tracheo-bronchitis (infective).

This disease, because of its prevalence and very high mortality rate, deserves real respect. It resembles laryngeal diphtheria, but is usually caused by the streptococcus or staphylococcus.

Tracheotomy is essential in the severe cases; suction through the canula is a very important part of the treatment.

Mechanical obstruction of the airways, even after tracheotomy, is of much more importance than the type of causative organism or its virulence.

After recognizing the disease as a clinical entity in 1920, and after having used all known treatments, our mortality rate in tracheotomized cases is still 60 per cent.

CONCLUSION

Cooperation, closer and more generally, of the general physician, internist and pediatrician with the bronchoscopist in the diagnosis and treatment of acute obstructions, inflammations and infections of the respiratory tract, will surely react to the benefit of all concerned, but especially to the benefit of infants and young children who are so prone to suffer seriously from acute involvements of the chest.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, April 3, 1939

THE PRESIDENT, DR. THOMAS W. LEWIS, IN THE CHAIR

Nasopharyngeal Fibromas

STANTON A. FRIEDBERG, M.D.

(Author's Abstract)

Three cases of nasopharyngeal fibroma are reported which have been successfully treated by a combination of irradiation and surgical diathermy. An attempt was made to analyze the histological alterations resulting from irradiation in these three cases. Despite the absence of significant histologic changes following irradiation in two of the three cases reported, a very definite decrease in the vascularity of irradiated areas was noted clinically. This clinical alteration greatly facilitated subsequent removal of tumor tissue. The validity of this observation was borne out by an attempt to remove a recurrence of tumor in the maxillary antrum. No irradiation had been used and an almost uncontrollable hemorrhage resulted. Because of the attachment of these tumors to the bones of the base of the skull and the involvement of these bones by pressure necrosis, it is suggested that the use of the cold snare and avulsion methods of removal be abandoned. The combination of irradiation and surgical diathermy offer a safe and effective method of treatment of vascular fibromas of the nasopharynx.

DISCUSSION

DR. T. C. GALLOWAY: Part of the incentive for this study came from reading of the treatment advised in a recent English text for nasopharyngeal fibroma. One is instructed to take powerful forceps to grasp the tumor directly and forcibly avulse it. Were

a tyro to follow this direction in one of the vascular types, he might find himself up to his knees in blood, figuratively speaking.

The tumors which are reported as occurring around adolescence and before are always vascular; later they seem to be hard fibromas. What happens? Do the patients die with vascular types if they are not removed, or is there an evolution of the tumor? I have removed, in older patients, three that were almost completely avascular with no recurrences, and I would suspect they might have been earlier in life overlooked angioid types.

The first cases were treated by radon implants for more than two years, then without any difficulty the main mass was removed by passing a snare around the pedicle and passing coagulating current through it. Such a tumor if removed completely gives little troublesome bleeding. If one cuts through the tumor and into the vascular spaces the bleeding may be terrific.

The recurrent tumor almost completely filled the antrum. It had been intended to ligate the internal maxillary artery transantrally, but the tumor overlay the artery which could not be exposed. Had I to do it again I would implant radon seeds and then coagulate later. The tumor bled very freely on attempted coagulation and after about two-thirds of the tumor was removed, the patient had lost about 700 cc. of blood, and I had to pack it and get out. On the third attempt I was able to ligate the internal maxillary artery, but the bleeding did not stop until the veins were also ligated. Incidentally, the ligation of the external carotid had little influence on bleeding, because of the collateral circulation. In the second case, after the radon was used, the tumor was coagulated and removed without difficulty. These tumors are clinically benign, but one should not depend upon irradiation alone for treatment, but should, I believe, complete the operation with surgical diathermy, otherwise there will be recurrence, as I had in my first case.

Dr. Louis T. Curry: Dr. Friedberg is to be commended for this concise and scientific presentation of this subject about which most of us know so little.

This type of fibroma is fortunately quite rare. In a large clinical experience at the Central Free Dispensary, the Cook County Hospital, and in the office of Dr. George Shambaugh, Sr., few of these vascular tumors have been studied and treated. The case presented by Dr. Friedberg is outstanding because of the size of the tumor, extensive pressure destruction and satisfactory results.

In dealing with these vascular fibromata, two features deserve special emphasis: 1. These tumors bleed freely and they should be easily diagnosed. Hemorrhage was profuse in my case following the slightest touch with a wet cotton applicator. He also bled spontaneously. The bleeding was not easily controlled. Examination was difficult because it was limited to inspection. Palpation of the growth was not feasible. A loose cocain pack aided diagnosis, but the tumor would not shrink sufficiently for proper examination because of its size. In spite of this condition, several attempts had recently been made to remove this polypus by an experienced otolaryngologist. 2. These tumors are difficult to remove. They are so inaccessible and yet the surgeon has his urge to remove them before they kill the patient. Extensive surgical resection was first considered in this patient. The most feasible route seemed through the antrum with ligation of the internal maxillary artery. This plan was abandoned because there was too much growth in the nasal cavity. Lateral pharyngotomy and splitting the hard and soft palate were also considered but discarded as too radical. Case reports where radium had been employed were discouraging because results were slow and the fibromata were not destroyed, merely retarded.

The vascular tumor in my case demanded emergency measures because of the invaded sphenoid. Radium was employed to control the hemorrhage and it was hoped there would be some shrinkage of the mass to facilitate better examination. Credit and thanks must be given to Dr. Thomas C. Galloway and Dr. H. A. Davis for valuable advice and helpful suggestions on the dosage for radium.

Dr. Friedberg has mentioned the difference in the cells of the tumors after treatment. Dr. Galloway's patients were younger and the cells did not show any tendency to fibrosis after treatment. My patient was near the age when regressive changes may take place. Irradiation may have hastened this change.

In closing, it should be stated that this man has complained for the past two months of difficulty in swallowing. There has been no loss of weight and chest examination reveals no abnormality. His dysphagia is worse at noon and in the evening. He has been more subject to head colds and pharyngitis with his atrophic nasal passages. Examination is easy but the ultimate prognosis remains to be seen.

Dr. R. H. Good: I think we are all agreed that the surgical method of removing nasal fibromas is very unsuccessful, and unless the base from which the tumor is growing is destroyed the tumor

will recur. My last case, about two and a half years ago, had a fibroma which extended to the level of the palate and it grew from the sphenoid base. The base of the tumor was about the size of a nickel. I should like to emphasize that all the treatment of the tumor mass, with the needle or forceps or snares, is not going to cure the tumor. It is the base that must be dealt with. If the base is destroyed the tumor will disappear.

In this last case I decided to use diathermy and not touch the tumor but destroy the base up to the bone, and in about three sittings I got around this base, and to my surprise a few weeks later I could not see any tumor, but had a space of bare bone the size of a nickel. Had I had less experience I would have been frightened, but this healed over, and the boy is well. If you treat the base with radium, or diathermy, you kill the base and the supply of blood to the tumor is destroyed and there will be less hemorrhage. This, of course, does not apply to large extensive tumors.

Dr. Frank Novak: I should like to discuss the pathology of these tumors. The pictures showed that these tumors in the young, 12 to 14 years of age, are highly cellular. The ones that occur later in life, at 20 to 25 years, are generally acellular, or more or less so. The hope for results with irradiation is to produce in the young the kind of growth which naturally occurs in those a little older—in other words, to produce a tumor which is less cellular and contains more fibers—a sclerosis. I have the feeling that if radiation is extensive enough and is carried on for a considerable time, and then time allowed to elapse, sclerosis does occur, and any kind of operation—not only diathermy, which is undoubtedly the best approach, but even a cutting operation—can be carried out with as much safety as in the individual of 24 or 25 years of age.

The designation angiofibroma, I believe, is a misnomer. It is highly vascular, it is true. You saw the large spaces filled with blood, but they are dilated capillaries. These are about 200 microns in diameter, but this distension is simply a distension of the capillaries and is the result of back pressure, passive congestion. These should not be called angiofibroma, because the neoplastic process is limited to the fibrous tissue and the blood vessels are not neoplastic. If we use the term angioma at all, we should use it as angiomatous or angiomatoid, because they are not true angiomas. There are many who believe that these tumors stem from the nerve tissues. We must consider that some at least emanate from nervous tissue.

Dr. Francis Lederer: Dr. Friedberg makes a very definite statement as to the attack on these tumors, which is to be desired,

yet from a practical viewpoint is not possible. These cases are individual cases and are determined by the site and size of the tumor. They seemingly arise mostly from the base of the sphenoid. As to the approach to the base, I cannot be impressed by Dr. Good's idea as to attacking the base, because as I remember a number of cases we have seen, the approach to the base would be scarcely possible. I believe the idea is good if that were possible. The effect of X-ray and radium on these tumors is not as stated, in our experience. As a matter of fact, there is something very contrary in the study of these tumors, namely, as a rule, when you disturb the tumor by radon you will find, instead of a decreased number of blood vessels, an increased number, therefore you can find even greater vascularity after irradiation treatment.

I recall one young man in our clinic who had been treated elsewhere with x-radiation of over 11,000 r. units. When operation was performed, despite a common carotid ligation, he bled just as freely as those who had not had previous radiation. Therefore I rise in defense of the evulsion technic. It is practical. This phase was handled with large epiglottis forceps. Dr. Fox gave blood transfusions to the boy each time and built him up, and again used the evulsion technic. I had a fatality from hemorrhage on the table in an instance where previous irradiation had been given. I obtained an autopsy and found a bony pedicle arising from the posterior wall of the sphenoid sinus, from which the tumor originated. In another case, by pressure the tumor forced itself through the sinus into the cranial cavity. All of this in spite of massive irradiation. I am sorry to have to think that the problem is still unsolved, and that I think each is an individual problem.

DR. R. H. GOOD: I have made diathermy applicators about eight or nine inches long, and they can be bent into different shapes. If I cannot go through the tumor I go around it, but I endeavor to get to the base, not paying any attention to the mass itself.

DR. M. REESE GUTTMAN: During the past thirteen years in my association with Dr. Joseph C. Beck, I have had occasion to see three of these cases. I believe that Dr. Friedberg is to be congratulated in stressing the histologic picture which is of real practical importance, especially from the clinical and therapeutic point of view.

Unfortunately, the literature has laid little stress on the presence of these thin-walled vascular spaces. These tumors should be labled as angiomatous fibromatas or angio fibromatas.

Our three cases responded well to irradiation. The first seen by us in 1926 refused surgical excision with diathermia, which Dr. Beck indicated at that time. The patient was referred to Dr. Orndorf for X-ray therapy, and the tumor disappeared in a short period of time. Six years later there was a recurrence and radon seeds were introduced into the mass, which disappeared, and the patient has remained well to date. Two other cases subsequently seen were also treated by the introduction of radon seeds. These also have regressed and there has been no recurrence to date.

The response to irradiation may give us a clue as to the histologic nature of these neoplasms. It is well known that fibromas are highly radio resistant, while angiomatas are more responsive to the gamma ray. The relative radio sensitivity that these tumors exhibit clinically might well indicate that the angiomatous portion of the histologic picture is probably more important, at least from the clinical and therapeutic point of view.

Dr. Howard C. Ballenger: The evulsion technic of removing hard avascular fibromas in older individuals should be defended, particularly in cases with a pedicle attachment to the body of the sphenoid bone, and with finger-like prolongations into the nasal cavities. In this type of growth it is frequently impossible to surround the mass with a snare, especially so if adhesions to the pharynx or palate are present, as they frequently are. Difficulty may be experienced in passing a diathermic electrode to the base of the tumor as wall. In these selected cases the evulsion technic may be the only practical method of removing the mass of the growth so that access may be had to the pedicled base. I am in accord with Dr. Friedberg that all these growths should have a preliminary irradiation.

Dr. T. C. Galloway: I would like to say to Dr. Lederer that it was obvious in these two cases that the radon seeds had a marked effect on the tumor. Before treatment one could not touch the second tumor with a forceps without getting a hemorrhage which took half an hour to control. After radon in this case it could be touched freely, manipulated, and the size was reduced four-fifths by the time diathermy was used. I was surprised that the vascular spaces were still so marked microscopically. These histologic sections represented perhaps recurrent tumor. It is my experience that the maximum effect of radon has taken place in a month or six weeks and after that time the tumors are growing again, and it is unwise to wait beyond the time you have maximum contraction for operation or using more radium.

Dr. Stanton A. Friedberg (closing): I should like to thank all the discussors. Dr. Galloway particularly emphasized the point that appeals to me in this connection, namely, that previously irradiated portions of these tumors, when they are subsequently attacked surgically, do not bleed as severely. In one boy the tumor recurrence in the antrum had not been irradiated and the bleeding was unusually severe. Dr. Curry has agreed that a marked difference in the ease of handling became apparent after radiation therapy. I think that is important. Dr. Good's point about dealing exclusively with the base of the tumor is excellent, but may be applicable to certain cases only. When these tumors are as large and extensive as were the three reported, and bleed so profusely, one is indeed confronted with quite a problem in attempting to gain access to the base alone.

With reference to Dr. Novak's discussion, I would like to believe that there is an age difference in the vascularity and cellularity of these tumors, but this does not seem to be the case. If anything, the tissue from our oldest patient was more cellular than the biopsies from the adolescent boys. Dr. Novak mentioned the desirability of simply awaiting regression after irradiation, to which I will reply that we are dealing with vital areas, and in one case invasion of the base of the brain was imminent. The terms "angiofibroma" and "vascular fibroma" are proposed solely in order to convey the difference in vascularity as contrasted with pure fibromatous tumors.

In answering Dr. Lederer, I can only add that we have been led to the clinical conclusion that irradiation very materially reduced the vascularity of these growths. Because these tumors originate in the periosteum at the base of the skull, traction and avulsion seem to me to entail considerable risk, and the operating room fatalities which he mentioned are exactly what we are endeavoring to avoid by proposing irradiation and surgical diathermy. Dr. Lederer also emphasized that irradiation therapy alone is not sufficient and in most cases this is undoubtedly true. Coagulating diathermy provides us with a safe method of dealing with the remainder of the tumor and controlling hemorrhage.

Dr. Guttman's results, using only radon implantation, illustrate that surgery may sometimes be unnecessary. Observation of the patients alone will enable us to judge what course to pursue. I believe that Dr. Ballenger's question regarding avulsion has been answered already.

Cerebral Edema as a Cause of Intracranial Hypertension of Otitic Origin

NORMAN A. LEVY, M.D.

(This paper is printed in full on page 999 of this issue.)

DISCUSSION

DR. NORMAN LESHIN: For some years we have been confronted with a clinical syndrome associated with otitis media, which Symmonds in 1931 suggested be termed "otitic hydrocephalus." The salient features of this syndrome were symptoms of increased intracranial pressure, namely, headache, vomiting and papilledema, without evidence of abscess formation, and with spontaneous recovery. The pathology was considered a hydrocephalus due to either excessive secretion from the choroid plexus, or defective absorption from the arachnoid villi.

This evening Dr. Levy has definitely shown that this clinical syndrome can be produced by cerebral edema, and that the pathology cannot be accurately determined unless thorough air studies are made. It is of great interest, further, to note in the literature that most of the cases reported where air studies were made revealed small ventricles instead of dilated ventricles as one would expect to find in cases of hydrocephalus. In spite of this finding, both the concept as well as the title of this syndrome has remained unchanged.

Now we are presented with a pathologic picture, cerebral edema, which can explain both the symptomatology and the pneumographic findings, as shown by Dr. Levy in this case report, with pneumographic studies and visual confirmation of the existence of edema when a cerebral decompression was done. It is peculiar, however, where subtemporal decompressions were done in a fair number of these cases, that none of the observers have mentioned the presence of cerebral edema. Davidoff and Dyke report having done a subtemporal decompression in thirteen of their fifteen cases of this type, without emphasizing cerebral edema. This may have been simply an oversight. On the other hand, it is still possible that this clinical syndrome may be produced by other types of pathology as a hydrocephalus, arachnoiditis, or even a posterior fossa lesion. With these possibilities in mind, and where the symptomatology may be fundamentally the same, it appears that air studies may be essential to determine the underlying pathology. In those cases where several spinal punctures clear up the condition, nothing further is required.

On the other hand, where the syndrome persists, further investigative measures are necessary as shown by Dr. Levy.

The causative factor for this condition is not necessarily an otitis media. Two cases followed a sore throat, and two others followed an upper respiratory infection. Some of the cases had a relapse following an upper respiratory infection. However, the large number of cases associated with lateral sinus involvement ranging from a phlebitis and perisinus abscess to thrombosis of the sinus must place the condition definitely as one of the otitic complications.

The therapy has been, essentially, repeated spinal drainage, and in obstinate cases, decompression. The papilledema gradually disappears over a period ranging from a few months to several years. Where the headaches return, they usually respond to spinal drainage alone.

Dr. Levy has shown the necessity for further study of this condition where both the etiology and the pathology are in doubt. Pneumographic studies seem to be a very definite step in this direction. Otologists who may come in contact with this condition should avail themselves of complete neurological co-operation in an attempt to solve this problem.

Dr. Levy has made a noteworthy contribution to this subject. He has shown the necessity of pneumographic studies, and his suggestion for changing the name of this syndrome should be seriously considered.

DR. HANS BRUNNER: The paper of Dr. Levy was very interesting, and I entirely agree with him that the conception of an "otitic hydrocephalus" as Symmonds has described it is not proven by the author of this idea. I personally have never been convinced by the deductions of Symmonds, although I have not been able to make an exact diagnosis in those cases of "otitic hydrocephalus" I have observed. Undoubtedly it portends great progress that Dr. Levy stressed the importance of encephalography in such cases. From the encephalography of his own case Dr. Levy draws the conclusion that he had to deal not with a hydrocephalus internus but with an edema of the brain, because the ventricles of the brain had a normal size and configuration, and because following the subtemporal decompression the brain was found moist and edematous. I cannot quite agree with the last conclusion. Provided that the ventriculography was performed prior to the subtemporal decompression, one should expect that the ventricles were narrower than normal in a case of edema of the brain. We must imagine that the brain, when

it suffers from an acute edema, becomes larger. By this increase of size it partly obliterates the arachnoidal spaces, but it also bulges out into the ventricles. Consequently the size of the ventricles should be expected to be narrower.

However, it is only of theoretical interest as to whether the interpretation of Dr. Levy is correct or not. From the practical viewpoint, of greater importance are two other questions: (1) How can these cases be differentiated from brain abscesses? (2) How should these cases be treated? In so far as the first question is concerned, three points seem to be of importance: (1) The cases of brain edema are usually children or juvenile individuals. (2) In cases of "brain edema" there can be frequently noticed a sudden development and a rapid increase of a choked disc, so that the papilla is elevated from five to six diopters within a few days. Such a rapid swelling of the papilla occurs neither in brain abscesses, nor, as a rule, in brain tumors. (3) Such patients do not give the impression that they are seriously ill, despite the marked changes within the eyegrounds, as already pointed out by Dr. Levy.

As far as the treatment is concerned, I have learned to be very conservative in these cases. When a mastoiditis is present, the simple mastoid operation is performed and the dura, particularly of the middle fossa, widely exposed. When there are signs of an infection of the sinus, both the sinus and the dura of the posterior fossa are exposed. A lumbar puncture is performed but not repeated too often. To date I have never been forced to do a further decompression of the brain. However, it is necessary that the physician should not become too apprehensive, since it sometimes takes many days for the choked disc to subside. The physician has to bear in mind that in these cases, as well as in brain abscesses, the choked disc seldom changes into a secondary atrophy of the optic nerve as it frequently does in brain tumors.

Dr. R. H. Good: Eighteen years ago I read a paper before this Society on the subject of brain abscess and brain irritation, and I showed then the symptoms of hypertension which arose from extradural irritation, either pus or toxin. No matter where this necrosis is, we get hypertensive symptoms. When we expose the dura it disappears, except when the abscess occurs over the lateral sinus. In this case, producing either a parasinusitis or a thrombosis, you see these symptoms of hypertension reappear and continue for a long time. I object to spinal punctures in the treatment of hypertension cases.

When I presented this paper Dr. Beck had just come back from the war, and he discussed it and said it was a wonderful pedagogical paper, but he did not think there was anything in the subject.

Dr. Joseph C. Beck: I want to recall to Dr. Good that when he presented that paper, I attacked it, and he should have given vent to his feelings against me, because Dr. Rogers who was associated with him in that paper was the surgeon. I certainly was opposed to the attitude these gentlemen took toward so many operations based on the symptoms alone and not findings. At that time x-ray was in its infancy and all one saw on the glass negative was a blur. We must not confuse the present time with the past in so far as the knowledge of this condition is concerned. I am sure the Society appreciates Dr. Levy's study of this one case and his references to the literature as a whole.

Dr. Norman Levy (closing): I wish to thank all the discussors. In reply to Dr. Smiley and Dr. Leshin, Davidoff and Dyke did not emphasize cerebral edema in their cases. They described it, but did not emphasize it. They found an abnormal amount of cerebrospinal fluid over the convexity, but they also found the underlying brain to be edematous. My idea is that it was the edema that was probably responsible for the small ventricles which they uniformly found.

In reply to Dr. Brunner, as to why the ventricles should not be smaller than usual. I cannot explain why; I can only state the findings. In our case the cerebral edema was not hypothetical, it was proven when decompression was done. I agree with everything Dr. Brunner said about differentiation from abscess. I think it is dangerous to wait too long under certain conditions in the presence of papilledema. One must test the visual acuity and the visual fields frequently, because there may be secondary changes to the optic disc and permanent change in the acuity. If visual acuity impairment is progressive, subtemporal decompression should be done, because a very rapid change can occur in the course of a week or ten days.

In regard to Dr. Good's objection to spinal puncture, I can only say that in the majority of cases this cures the patient and, inasmuch as no harm is done, I can see no objection.

Simplified Construction of Facial Moulds and Prosthesis

(Demonstration with colored movies and models)

SAMUEL PELUSE, M.D.

(Author's Abstract)

The technic of making facial moulds (life masks) as perfected by Dr. Fox and the essayist in the past two years was shown. The making of life masks is an essential adjunct to reconstructive surgery of the face and the construction of prosthesis. They are accurate three-dimensional reproductions of the patients' faces and lend themselves to preoperative study, measurement and planning.

The models demonstrated were of wax and rubber. Wax models are hard to handle and difficult to store and are made only when a rapid specimen is needed. Liquid latex rubber is ideal for making models, as the resultant product tolerates undue rough handling and will not deteriorate. Of interest are the reproductions of operated temporal bones. In two the exposed dura is represented and when palpated one gets the sensation of actually feeling the dura. These models lend themselves very well to teaching purposes.

DISCUSSION

DR. JOSEPH C. BECK: I want to compliment Dr. Lederer on having developed the method to this perfection. It would be too bad to let this demonstration go without discussion. I have had a good many years experience in making models for repair of defects about the face. We have done a good many things along this line of prosthesis with excellent results. This exhibit is wonderfully demonstrated, particularly the pathology of the tissues involved. One cannot get by the appearance what can be obtained by the feel; for instance in the temporal bone specimens to palpate the dura. These are very important, and are interesting from the standpoint of operators. We may also have gross specimens reproduced to show pathology. We are very short of gross pathologic material in the registry in Washington and this is going to fill a great need in working out these things.

Dr. Peluse is to be congratulated on this work, which is much needed by otolaryngologists and men who are interested in radical exenteration of sections of the face in malignancy or trauma, and can have something to show the patient as to the end result, when so much tissue has to be sacrificed.

DR. FRANCIS LEDERER: I rise to say that I cannot accept credit for this presentation. The work was done by Drs. Fox and Peluse, with the help of Mrs. Peluse. My interest in this type of work goes back a number of years to prostheses (Arch. Otolaryngol., 8:531 [Nov.], 1928), as well as material for teaching. This work has filled a great need in our department in this regard. Dr. Peluse is now making it a part of clinical teaching. You may feel that it is purely a technician's work, but I think it is important that we create an interest in this type of work, because it is an essential part of our specialty, not only from the teaching point of view, but also in the replacement of gross defects. The evening devoted to this was so short that many details were left out, but I am quite sure the exhibit is well worth study. Aside from its clinical application it is economical. The models are reproduced at small cost, and are not easily broken, and can be preserved.

Abstracts of Current Articles

NOSE

Nasal Allergy in Children.

Barnett, E. J., and Carnahan, H. D. (Spokane), Arch. Otolaryng. (Aug.), 1939.

It has been reported that 44 per cent of the patients with chronic nasal complaints seen in routine otolaryngological practice have nasal allergy. Both rhinologists and pediatrists must become allergic-minded to arrive at an accurate diagnosis and to obtain better results in the management and treatment of many nasal complaints in children.

The conclusions drawn by the authors are as follows: (1) Allergy should always be suspected as the cause of rhinitis in children. (2) Cytologic examinations of the nasal secretions with predominance of eosinophils offer confirmatory evidence. (3) Routine repeated cytologic examinations for all patients with chronic nasal symptoms are important. (4) Local treatment for nasal allergy must be conservative. (5) Disappointments in rhinologic practice often result in failure to recognize and relieve associated allergy. (6) Prophylactic allergic measures are often sufficient to give relief from nasal allergy. (7) Allergic testing and hyposensitization may be reserved for unrelieved or complicated conditions. (8) The importance of allergy to the rhinologist cannot be overemphasized.

TOBEY.

Atrophic Rhinitis: Treatment with Estrogenic Substances, With Biopsy Before and After Treatment.

Eagle, Watt W.; Baker, Roger D., and Hamblen, E. C. (Durham, N. C.), Arch. Otolaryng. (Sept.), 1939.

An extensive review of the literature dealing with estrogenic substances for the treatment of atrophic rhinitis is presented. There are 22 cases reviewed, which were treated with estrogenic substances.

Biopsy specimens were obtained from 14 of these 22 from the middle turbinate before, during, and after treatment of these cases. Twenty-one of the 22 patients treated with estrogenic substances for atrophic rhinitis were clinically improved both subjectively and objectively and desired to continue treatment. However, in the 14 cases which were biopsied, no obvious morphologic change had taken place following the treatment.

TOBEY.

PHARYNX

Mixed Tumors of the Salivary Gland Type Seen in the Pharynx.

Fox, C. Calvin (Philadelphia), Arch. Otolaryng. (July), 1939.

Mixed tumors of the salivary gland type occur entirely separated from the glands, are benign and can occur at any age in the neck and pharynx.

They probably originate from embryonic cells that become separated from the body of the gland. They contain tissues derived from both the ectoderm and the mesoderm. The microscopic structure is varied and complex.

They develop gradually in the tonsillar and the palatine area until they occupy a large portion of the pharyngeal space.

They are firm, slightly nodular and completely encapsulated, do not break down and cause little local reaction or discomfort.

Their appearance and behavior are so distinctive that they can readily be diagnosed. Biopsy is unnecessary and contraindicated, because taking the specimen complicates removal of the tumor.

Local applications and roentgen or radium irradiations are of no value in treating these tumors.

Surgical removal through an intrapharyngeal incision is the most satisfactory way to eliminate the tumor.

Care should be taken to preserve the continuity of the capsule. If it is ruptured, a piece of tumor tissue may remain and grow again.

There is little postoperative reaction; recovery is rapid, and function is soon restored.

There will be no recurrence if all the tumor tissue is removed.

TOBEY.

LARYNX

The Larynx of the Tuberculous Child.

Rubin, Herman, and Galburt, Samuel (Brooklyn, N. Y.), Arch. Otolaryng. (Sept.), 1939.

Few tubercular children are seen by the laryngologist for laryngeal examination because the early symptoms are mild and are overshadowed by the constitutional signs. Routine examination of

the larynx of all children with pulmonary tuberculosis should be done. Such examination would disclose a large number with laryngeal involvement.

In this report 100 tuberculous children were examined by indirect laryngoscopy. Changes in the larynx were found in 23 of 54 children with tuberculosis whose sputum, gastric content and fæces were negative for the bacilli. There were 21 normal larynges found among 46 children whose sputum, gastric contents or fæces were positive. Bronchoscopic examination should be performed on tuberculous children who have negative sputums because a direct smear will often show the presence of tubercle bacilli. It is contraindicated when active laryngeal lesions exist.

The chief therapeutic measures are vocal rest and the application of galvano-cautery electrodes. The general and pulmonary condition must also be treated. Collapse therapy has greatly improved the local condition and the general prognosis.

TOBEY.

EAR

Labyrinthine Fistulas: Report of Experiments on the Vital Responses to Various Methods of Producing Defects in Bone.

Canfield, Norton (New Haven), Arch. Otolaryng., (July), 1939.

Dr. Canfield reviews the work to date on methods of making and maintaining fistulas of the labyrinth. It is felt now that the technical problem of making the fistula has been satisfactorily solved, but the method of maintaining a patent fistula is still debatable. The work of Holmgren and Lempert shows their method of fistulization of the external canal to be adequate, although their technique in making the fistula differs.

The author has done experimental work on animals to determine the difference in the bone of the semicircular canal immediately after the fistula had been made by the scraping method of Holmgren and also by the burring method of Lempert. The questions Dr. Canfield wishes to finally answer are: (1) Has the bone been affected differently by the two methods? (2) Has the actual mechanical process changed the bone? (3) Should heat produced by the friction of the drill be considered? (4) Does the drill injure bone cells which are not removed? (5) Has the bone's power of regeneration been diminished?

The present experimental work is not finished. However, the conclusions drawn from the work done are: (1) Morphological variations exist in bone fistulas produced by different methods. (2) The response of vital periosteal bone differs significantly, when defects are made with different instruments, although the defects are uniformly covered by the same type of tissue.

TOBEY.

Otitic Sinus Thrombosis Causing Intracranial Hypertension.

Gardner, W. James (Cleveland), Arch. Otolaryng. (Aug.), 1939.

Dr. Gardner attempts to explain the syndrome of increased intracranial pressure, which occasionally accompanies an attack of otitis media and which is not due to meningitis, encephalitis, or abscess of the brain. It is thought that this syndrome of increased intracranial pressure with the localized signs occurring after an attack of otitis media is usually due to sinus thrombosis.

It is felt that in the past, false interpretation of observations have been made on cases of this type. First, the large amount of cerebrospinal fluid obtained at lumbar puncture before the pressure is reduced to normal does not indicate an increase in the amount of spinal fluid present. Because the venous outflow is obstructing, the spinal fluid removed is immediately replaced by an increase of venous blood within the already engorged cerebral veins. This engorgement of the veins forces a larger quantity of cerebrospinal fluid out of the lumbar puncture needle than one would be able to obtain if the venous obstruction were not present.

Second, when trephination is performed, the exposed portion of the arachnoid membrane is found to be distended with fluid. This observation has led the surgeon to assume that external hydrocephalus is present. In most cases, this is not true, since encephalograms in these cases are essentially normal. It seems fair to assume that the subarachnoid space is distended as disclosed at trephination because the release of pressure at the site of operation produces local accumulation of fluid in the subarachnoid space in this area.

Third, another observation which the author feels to be false is the belief that hydrocephalus is present in these cases because of the copious flow of fluid that occasionally occurs when the cannula is introduced into the lateral ventricles. This has been interpreted as meaning that the ventricles are dilated. This assumption is incorrect as shown by encephalographic examination, which demonstrates that in these cases the ventricles are normal in size or even smaller

than normal. When a large quantity of fluid is obtained from the ventricle in a case of this sort, it means that since the cerebrospinal fluid system is not obstructed, the expanding intracranial veins squeeze all the fluid toward the point of exit. In these cases, one cannot estimate the size of the ventricles from the amount of fluid obtained by ventricular puncture.

In conclusion, the author feels that instead of referring to the syndrome by a variety of names, it appears preferable to adopt a descriptive title, such as "sinus thrombosis causing intracranial hypertension."

TOBEY.

Otitic Infections Due to the Pneumococcus Type 3.

Maybaum, J. L., and Druss, J. G. (New York), Arch. Otolaryng. (July), 1939.

This paper is an analytical study of 73 cases of pneumococcus, type 3 infection of the ear, taking into consideration the clinical course, pathology, diagnosis, treatment and final result. The cases studied are classified according to the clinical course and the otoscopic picture.

Early examination of the type of organism in the discharge from the middle ear or the mastoid is of great importance. Extensive destruction through the temporal bone is a characteristic observation made at operation or on histological examinations. These marked changes are frequently encountered in spite of latent innocuous clinical courses.

Intracranial complications are associated more commonly with otitic infections due to this organism than with those due to streptococcus. These complications usually occur late in the disease, but occasionally are found in the early stages.

The following points of treatment are stressed: (1) Early paracentesis to establish a diagnosis by a culture report; (2) an autogenous vaccine should be made and used as soon as the organism is identified; (3) if operative work is necessary, complete exenteration of all diseased, bony structures should be done with special investigation of the various routes to the petrosa. If petrous involvement occurs, early operative intervention is demanded. The administration of serum and sulfapyridine should be employed; (4) there is no specific therapy for pneumococcus, type 3; (5) prolonged observation of the patient after healing of the mastoid is of extreme importance.

TOBEY.

The Eustachian Tube: Abnormal Patency and Normay Physiologic State.

Perlman, H. B. (Chicago), Arch. Otolaryng. (Aug.), 1939.

An attempt is made to correlate all the work done in the past 100 years on the eustachian tube, in an effort to develop a rational basis for a functional test. Emphasis is laid on the simple, practical method for a quantitative determination of the patency of the eustachian tube by the use of the mercury manometer of the common blood pressure apparatus. This method is described in detail and illustrated by drawings. By this method, it was found that the eustachian tube offers a minimal resistance of about 20 mm. of mercury when the patient is in the erect position. However, a resistance of up to 60 appears to be compatible with normal auditory function. It has been found that the resistance of the tube is regularly decreased by exercise and the resistance is greatly increased by assuming the prone position and flexing the neck toward the chest. Unless quantitative studies such as the above are made the pathologically patent tube is likely to be overlooked.

When the tube opens to pressure below 20 mm. of mercury, it may be considered pathologically patent. In such cases, even the small pressures developed in respiration and phonation are sufficient to open the tube.

The author lists a number of factors which have been found to result in closure or in opening of a pathologically patent tube. The study of a group of cases here presented indicates that a lack of tonus of the muscle that affects the eustachian tube (tensor veli palatini) that is supplied by the fifth nerve is the principal factor in producing this clinical entity.

TOBEY.

MISCELLANEOUS

Respiratory Changes in Vitro in Normal and Malignant Tissues Following Irradiation.

Goldfeder, Anna, D.Sc., M.U.C. (New York), Am. J. Cancer, 36:603 (Aug.), 1939.

From a series of tissue culture experiments using normal malignant mammalian tissue as explants, the author concluded that the respiratory processes of these tissues are much less vulnerable to a given dose of radiation than the proliferative processes. With unfiltered radon, for instance, 2200 mc. hours entirely inhibited the proliferation of explants, while 6710 mc. hours reduced oxygen con-

sumption only 64 per cent. The effective dose of radiation (radium and x-ray) was shown to depend on the type of rays and the dose administered to these explants. The effective dose in vitro is far in excess of that which can be applied to these animals (mice) in vivo. Thus the effect of therapeutic doses of radiation on tumors in vivo must be an indirect one, possibly through the more sensitive blood and lymph channels, cutting off the nutritive supply to the tumor.

JORSTAD.

Further Experience with Roentgen Therapy for Bronchiectasis.

Berck, M., M.D., and Harris, W., M.D. (New York), Radiology, 32:693 (June), 1939.

In a group of cases observed over a period of one to six years, roentgen therapy in moderate doses was used as the sole method of treatment for chronic suppurative bronchiectasis. Symptomatic improvement occurred in a considerable proportion of cases. Some cases appear clinically well, with practically complete cessation of the symptoms of expectoration and cough. No recurrence of symptoms has been noted in these, in some instances for a period of six years.

JORSTAD.

Pulmonary Asbestosis. A Report of Bronchial Carcinoma and Epithelial Metaplasia.

Lynch, K. M., M.D., and Smith, W. A., M.D., Am. J. Cancer, 36:567 (Aug.), 1939.

The authors have encountered seven cases of primary lung carcinoma in 2,343 consecutive autopsies over the past twelve years. Thirty-five cases showed some degree of asbestosis. Incidence of primary pulmonary carcinoma in the series is 0.3 per cent. There were two cases of pulmonary carcinoma in the asbestosis group, or an incidence in this small series of approximately 6 per cent.

In the advanced asbestosis case, the bronchial epithelium showed a metaplasia.

JORSTAD.

Books Received

Cancer of the Larynx.

By Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., Honorary Professor of Broncho-Esophagology and Consultant in Broncho-Esophagologic Research, Temple University Medical School, Philadelphia; and Chevalier L. Jackson, A.B., M.D., M.Sc. (Med.), F.A.C.S., Professor of Broncho-Esophagology, Temple University Medical School, Philadelphia. Three hundred and nine pages with 189 illustrations on 116 figures, and five plates in colors, containing 50 illustrations. W. B. Saunders Company, Philadelphia and London, 1939. Cloth, $8.00.

Here the Jacksons, father and son, have gathered the fruit of their wide experience and placed it between covers for the instruction of the laryngologist.

The subject has been approached from three angles: (1) Procedures; (2) General Considerations, and (3) Historical Notes; this, as the authors point out in their preface, to meet all tastes and requirements. The first division presents its material point blank as a working manual for the practitioner faced with a patient and seeking ways and means. The second is intended to instruct him in his more absorptive moments when there is time for reflective consideration of sources and opinions. The historical notes are "for lighter reading; perhaps in his study, or on a train."

Throughout the work, descriptions are concise and illustrations apt, and the methodical approach which characterizes the clinical work of these authors prevades the book. Textbook style is maintained with a liberal sprinkling of italics, so that the reader retains the impression of the systematic outline which underlies the text.

Cancer Laringeo Su Tratamiento Quirurgico (Cancer of the Larynx—Its Surgical Treatment).

Dr. Ricardo H. Bisi, Ex interno del Hospital Nacional de Clinicas, Medico del Servicio de Oto-rino-laringologia de los Hospitales Nacional de Clinicas y Alvear. Paper. Royal 8 vo. of 355 pages with 100 illustrations. Buenos Aires: El Ateneo: 1938. (In Spanish.)

For the edification of anyone reading the language, this work affords an interesting comparison with the foregoing and, in the manner of presentation, the two texts have much in common. The sources quoted here are predominantly European and the author's

thought reflects them to a large extent. That two such definitive works on the subject of laryngeal cancer should have appeared within recent months is evidence that the contemporary viewpoint has, in a measure, crystallized and that laryngologists at large can treat cancer of the larynx with some confidence in their choice of method.

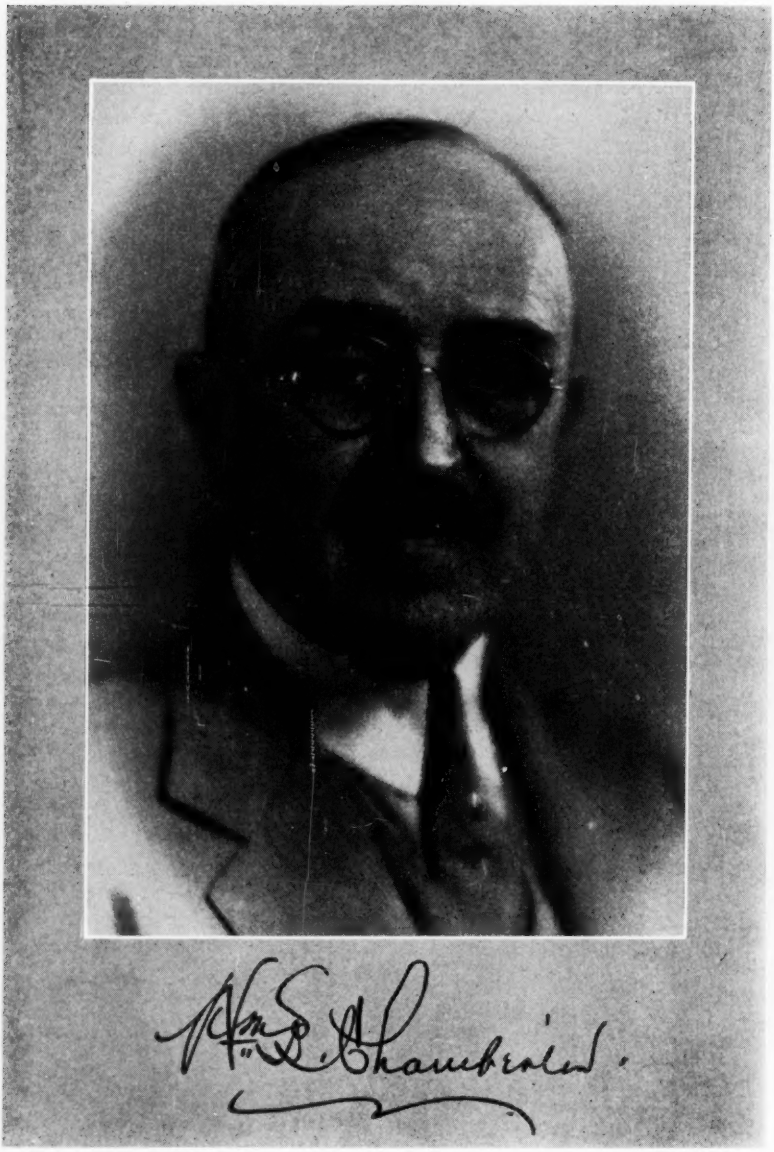
The Diagnosis and Treatment of Diseases of the Esophagus.

By Porter P. Vinson, B.S., M.A., M.D., D.Sc., F.A.C.P., Professor of Bronchoscopy, Esophagoscopy and Gastroscopy, Medical College of Virginia, Richmond, Virginia. Charles C. Thomas, Publisher, Springfield, Illinois, and Baltimore, Maryland.

A small, reasonably complete and very practical manual of esophageal disorders. The work is well documented. Perusal of the bibliography suggests that much of the experience upon which these pages rest is personal, as the author has contributed widely to the literature of the subject.

Diseases of the Nose and Throat.

Charles J. Imperatori, M.D., F.A.C.S., Professor of Otolaryngology, New York Polyclinic Medical School and Hospital; and Herman J. Burman, M.D., F.A.C.S., Adjunct Professor of Otolaryngology, New York Polyclinic Medical School and Hospital. Cloth. 8 vo. of 726 pages with 480 illustrations. Second Edition Revised. J. B. Lippincott Company, Philadelphia, 1939. Price, $7.00.


Wm. E. Chamberlain.

WILLIAM B. CHAMBERLIN

1873-1939

September 6, 1939, brought to the members of the various Otolaryngological Societies a great loss in the death of Dr. William Bricker Chamberlin of Cleveland, Ohio.

Dr. Chamberlin was born in Ohio, was educated at Oberlin College in Ohio, received his professional training at Western Reserve University in Ohio, and practiced his specialty at Cleveland, Ohio. The sense of loss brought by his passing is, therefore, most keenly felt in his native state where he was honored, respected and loved as a physician, teacher and friend.

But the sense of loss knows no state boundaries, for Dr. Chamberlin was known throughout the country as a skilled otolaryngologist. He had been president of national and local associations, the American Laryngological Association (1937), the American Bronchoscopic Association (1925) and the Cleveland Academy of Medicine. He was a Fellow of the American College of Surgeons.

He was vitally interested in those who were handicapped by otolaryngological diseases and expressed that interest in particular through his work for the Cleveland Society for the Hard of Hearing, of which he was president for more than ten years.

To his confreres Dr. Chamberlin was known as a man of excellent judgment. His frankness was sometimes startling, but he was alway honest and sincere in his dealings with his patients and with his colleagues. He will be greatly missed in the deliberations of the societies of which he was an honored member.

To his patients he was not only a physician but a friend upon whose counsels they relied and to whom they appealed with confidence in his ability to help them.

To his friends he was loyal, sincere and thoughtful of their interests and they, as well as his colleagues, esteemed it a privilege to know him.

Through these pages the national Societies of Otolaryngology express to his wife and two sons sincerest sympathy in their great loss which is shared by the friends and colleagues of Dr. Chamberlin.

JUSTIN M. WAUGH, M.D.

PUBLICATIONS OF DR. WM. B. CHAMBERLIN

The Permanent Closure of Large Perforations in the Tympanic Membrane with Marked Increase in the Hearing Distance. Cleveland Medical Journal, 7:342, 1908.

Experimental Nystagmus and an Application of its Principles to a Diagnosis of Lesions of the Inner Ear and Cerebellum. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (March), 1909. Candidate's thesis, American L. R. & O. Society.

Nystagmus with Relation to Diagnosis of Lesions of the Inner Ear and Cerebellum. Interstate Medical Journal (June), 1909.

Two Cases of Locomotor Ataxia with Abductor Paralysis of the Vocal Cords. Cleveland Medical Journal, 8:281, 1909.

The Tonsil Question. Interstate Medical Journal. Review of Literature (Oct.), 1909.

Tonsil Enucleation with Capsule with Description of Technique. Cleveland Medical Journal, 8:403, 1909. Read before the Clinical and Pathological Section of the Academy of Medicine of Cleveland (April 9), 1909.

Vaccine Therapy in Otology. Review of Literature. Interstate Medical Journal (Jan.), 1910.

Some Practical Points in the Extirpation of the Tonsils from an Experience of Five Hundred Cases. The Laryngoscope (Sept.), 1910. Read before the Middle Section, American R. L. & O. Society, Detroit, Michigan, Feb., 1910.

Adenoids in Infancy. Review of Literature. Interstate Medical Journal (June) 1910.

A Brief Review of Some Recent Advances in the Treatment of Pathological Conditions of the Nose. Cleveland Medical Journal (July), 1910. Read before the Jefferson County Medical Society.

Nystagmus as Related to Diseases of the Inner Ear and Cerebellum. Ohio State Medical Journal (Aug.), 1910. Read before the Nose, Throat and Ear Section of the Ohio State Medical Assoc., 1910.

Non-Suppurative Ethmoiditis. Review of Literature. Interstate Medical Journal (Nov.), 1910.

Fibroma of the Naso-Pharynx. Review of Literature. Interstate Medical Journal (March), 1911.

Fibroma of the Nasopharynx with Report of Four Cases. Read before the American Rhin., Laryng. & Oto. Society, Atlantic City, May, 1911. Published in their Transactions and in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1911.

Nasal Tuberculosis with Report of Two Cases. Ohio State Medical Journal (Aug.), 1911. Interstate Medical Journal (July), 1911. Read before the Nose, Throat and Ear Section, Ohio State Medical Assoc., 1911.

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Epidemic of Sore Throat. Review of Literature. Interstate Medical Journal (May), 1912.

Hexamethlanamin: Its Use in Diseases of the Upper Air Passages. Review of Literature. Interstate Medical Journal (Oct.), 1912.

The Relation of Bronchial Asthma of Pathological Conditions of the Nose. Review of Literature. Interstate Medical Journal (April), 1913.

Perforation of the Nasal Septum. Ohio State Medical Journal (April), 1913. Cleveland Medical Journal, 1912. Read before the Nose, Throat and Ear Section, Ohio State Medical Assoc., Dayton, 1912.

The Catarrhal and Suppurative Diseases of the Accessory Sinuses of the Nose by Ross Hall Skillern. Book Review. Cleveland Medical Journal (July), 1913. Interstate Medical Journal (Aug.), 1913.

The Labyrinth by Alfred Braun. Book Review. Interstate Medical Journal (July), 1913. Cleveland Medical Journal (July), 1913.

The Prevention of Speech Defects in Children. Review of Literature. Interstate Medical Journal (Oct.), 1913.

Report of Case of Nasal Polypi Involving the Orbit, Frontal Sinus and Anterior Fossa of the Skull. The Laryngoscope (Nov.), 1913. Read before the American L. R. & O. Society, Washington, May, 1913, and published in their transactions.

Diseases of the Ear, by Philip D. Kerrison. Book Review. Interstate Medical Journal, 1913.

The Alcoholic Carbolic Injection of the Spheno-palatine Ganglion. A Review of the Literature. Interstate Medical Journal (Jan.), 1914.

A Manual of Diseases of the Nose and Throat, by Cornelius G. Coakley. Book Review. Interstate Medical Journal (Nov.), 1914.

Diseases of the Nose, Throat and Ear: Medical and Surgical, by Wm. Lincoln Ballener. Book Review. Interstate Medical Journal (Nov.), 1914.

Suspension Laryngoscopy. Review of Literature. Interstate Medical Journal (Dec.), 1914.

A Textbook of the Diseases of the Nose and Throat, by B. Braden Kyle. Book Review. Interstate Medical Journal (Oct.), 1915. Third edition.

The Endonasal Operation of the Lachrymal Sac: Review of Recent Literature. Interstate Medical Journal (Feb.), 1916.

Cysts of the Larynx. American L. R. & O. Society, White Sulphur Springs, May, 1916.

Suspension Laryngoscopy—An Aid in Removing Foregin Bodies from the Upper Larynx and Esophagus. Report of Cases. Thesis. American Laryngological, 1917. Published in their Transactions.

Septic Sore Throat. Cleveland Medical Journal (April), 1917.

An Epidemic of Sore Throat Due to Milk. Cleveland Medical Journal (April), 1917.

Sphenoid Sinus. Present-Day Value of Surgical Procedure. Cleveland Medical Journal (Mar.), 1917.

Skin Grafting in Mastoid Operations. Cleveland Medical Journal (April), 1917.

Medical Publicity. Editorial. Cleveland Medical Journal (May), 1917.

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Personal Experiences in Bronchoscopy and Esophagoscopy—Illustrated with Lantern Slides. Cleveland Academy of Medicine, Feb. 21, 1930.

Xerostomia—Is It a Deficiency Disease? American L. R. & O. Society, Atlantic City, N. J., May, 1930.

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Mucocele as a Cause of Proptosis: Report of Six Cases. Thayer L. Parry and Wm. B. Chamberlin. Presented at the annual meeting of the American Laryngological Assoc. in conjunction with the Congress of American Physicians, Washington, May 8, 1933. To be published in the Transactions of the American Laryngological Assoc. and in the Archives of Otolaryngology.

Is Swimming a Safe Form of Exercise or Sport? Report of a Few Fatalities Following Swimming. Read before the N. E. T. Section of the Academy of Medicine, Dec., 1933.

After 30 Years the Doctor Looks at Himself and His Profession. Read before the Philosophic Club, Jan. 23, 1934.

Rhinoscleroma—Is It an Indigenous Disease? Read before the American Laryngological Assoc., May 31, 1935. Published, Archives of Otolaryngology and Transactions of Amer. Laryngological Assoc.

Removal of Molar Tooth from Left Main Stem Bronchus. Read before the American Bronchoscopic Society, June 1, 1935. Published in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY.

Osteoma of the Nasal Accessory Sinuses, with Report of Cases. Discussion of Paper by Dr. Carmody before the American L. R. & O. Society, June 5, 1935.

Suggested Routine Technique for Emergency Tracheotomy. Read before the Laryngological Association, June, 1936. Published in the Laryngoscope (Oct.), 1936.

Some Practical Considerations of Conditions in Otolaryngology of Interest and Importance to the General Practitioner. Read before the Ohio State Medical Association General Meeting, April 29, 1937. Published in the Ohio State Medical Journal.

Address of the President of the American Laryngological Assoc., Atlantic City, N. J., May 31, 1937. Published in the Transactions of the Society.

The Preventible Deafness of Childhood. Radio Talk, WJAY, Feb. 11, 1937. Published in the Ohio State Medical Journal (Feb.), 1937. Review in Archives of Pediatrics (March), 1937.

The Germans of Pennsylvania—More Commonly Known as the "Pennsylvania Dutch." Read before the Philosophic Club, Cleveland, October, 1938. Also before the Pasteur Club and the Rowfant Club of Cleveland.

The Prevention of Septal Haematoma After Submucous Resection. Read before the American Laryngological Assoc. at Rye, N. Y., May 24, 1939.

ROBERT SONNENSCHEIN

1879-1939

Dr. Robert Sonnenschein was born in Chicago, July 10, 1879. He attended the grade and high schools. He graduated at Rush Medical in 1901 when he was twenty-two years of age. After completing his internship at Cook County Hospital he went to Europe where he pursued postgraduate study in diseases of the ear, nose and throat. He visited the clinics in Vienna, Berlin and Königsberg. In 1924 he was made Professor of Otolaryngology at the Post Graduate School and in 1926 Attending Otolaryngologist at the Michael Reese Hospital and also Instructor in Otolaryngology at Rush Medical College. In 1933 he was appointed assistant clinical Professor of Laryngology and Otology. He served as president of the Chicago Laryngological and Otological Society in 1922-23. He was a fellow of the American College of Surgeons and a member of the American Medical Association, Chicago Pathological Society, Chicago Institute of Medicine, Chicago Laryngological and Otological Society, American Academy of Ophthalmology and Oto-Laryngology, American Laryngological, Rhinological and Otological Society and other organizations.

His written works consist of a "Syllabus of Lectures on Nose and Throat," popular for many years among students at Rush Medical College; a chapter on testing of hearing in the Cyclopedia of Medicine, 1936; a chapter on hearing in Jackson and Coates, "The Nose, Throat and Ear and Their Diseases," published in 1939; a section on surgery of the ear in Christopher's "Text Book of Surgery," 1935. His "Reviews on the Functional Examination of Hearing" published in the Archives of Otolaryngology from 1925-1938 are a splendid contribution to the science of medical acoustics. He spent a great deal of time at the Riverbank Laboratories, Geneva, Illinois, where he worked in an advisory capacity and suggested the type of tuning fork most suitable for the use of otologists. He was especially interested in the aluminum-magnesium alloy fork manufactured at Riverbank.

Replete as his life was with medical interests, he participated in social and cultural activities and he enjoyed the admiration of a wide circle of friends. Sonnenschein was well regarded as a student of his subjects and he was skillful in diagnosis and technic. He had artistic tastes, he collected rare books, especially those referring to the history of his specialty. He also had interesting collections of

Mezzo tints, book plates and autographs. He was a kindly, friendly man and he returned with warmth and appreciation the loyalty and friendship of his colleagues and companions. His passing will be mourned by his host of friends who are better men and women for having known him.

IRA FRANK.

PUBLICATIONS OF DR. ROBERT SONNENSCHEIN

1907-1938

1. The Determination of a First Class Risk. Chicago M. Recorder (April), 1907.

2. Analysis of the Weber Test in 100 Cases. Laryngoscope (May), 1911.

3. Resonators, with Special Reference to the Schaefer Apparatus. Laryngoscope (May), 1913.

4. Pemphigus Involving Primarily the Mouth and Throat, with Report of Cases. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (Sept.), 1914.

5. A Suggestion Regarding the Rinne Test. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (June), 1916.

6. Primary Mastoiditis with Report of a Case. Illinois M. J. (Sept.), 1917.

7. Ear, Nose and Throat Involvement in the Recent Influenza Epidemic. International Clinics, 4:28, 1918.

8. Proper Diagnosis as a Guide to Prognosis and Operative Treatment of Impaired Hearing. Illinois M. J. (Dec.), 1918.

9. Some Interesting Ear Cases. M. Clin. North America (July), 1919.

10. Some Nonsuppurative Forms of Headache. M. Clin. North America (May), 1920.

11. Dermoids of the Anterior Mediastinum with Report of a Case. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (June), 1920.

12. Radium in the Treatment of Malignant Tumors of the Nose and Throat. J. A. M. A. (Sept. 25), 1920.

13. Headaches: with Special Reference to Those of Nasal Origin. Illinois M. J. (Oct.), 1920.

14. Four Nasal Cases and an Aortic Aneurysm with Laryngeal Symptoms. M. Clin. North America (July), 1921.

15. Resonators as Possible Aid in Tuning Fork Tests—A Preliminary Report. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (Sept.), 1921.

16. Anesthesia in Nose and Throat Work. J. A. M. A. (Oct.), 1921.

17. A Study of the Schwabach Test in One Hundred Cases. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (March), 1922.

18. A Study of the Rinne Test in One Hundred Cases. Laryngoscope (March), 1922.

19. Acute Mastoiditis and Some of Its Complications. M. Clin. North America (May), 1922.

20. Study of Reinforcement of Sound by Means of the Schaefer Resonators. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (March), 1923.

21. Use of Tuning Fork Stem for both Air and Bone Conduction in the Rinne Test. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (March), 1923.

22. Alcohol Injections as a Possible Adjunct to Tonsillectomy Under Local Anesthesia. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (Sept.), 1923.

23. A Group of Nose and Throat Cases Presenting General Systemic Symptoms. M. Clin. North America (Sept.), 1923.

24. Eine Untersuchung über die Schallverstärkung mittels des Schaeferschen Resonatoren-Apparates. Beitr. Anat., Phys., Path. & Ther. des Ohres, Nase, Halses, v. 19, Heft 3/5, 1923.

25. A Study with the Webster Phonometer of the Sounds Emitted by Different Parts of the Tuning Fork Prongs. Tr. Am. Otol. Soc., 1923-24.

26. Methods and Interpretation of the Fundamental Tests of Hearing. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (June), 1924.

27. Some Points in the Observation of Children Who Are Deaf or Hard of Hearing. Oralism and Auralism (July), 1924.

28. A Series of Unusual Ear, Nose and Throat Cases. M. Clin. North America, 1924.

29. Calcium and Parathyroid Glands in Relation to Hyperesthetic Rhinitis. J. A. M. A. (Dec. 20), 1924.

30. Studies of the Rinne Test with Special Reference to the Tuning Fork Stem for Both Bone and Air Conduction. Laryngoscope (Dec.), 1924.

31. Further Experiences with Dilute Alcohol Nerve Blocking Anesthesia in Tonsillectomy. Illinois M. J. (March), 1925.

32. Schaefer-Galton Whistle. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (Sept.), 1925.

33. Headaches from the Systemic and Cranial Standpoint. Wisconsin M. J. (Oct.), 1925.

34. Report of Committee on Standardization of Tuning Forks and Methods of Hearing Tests. Tr. Amer. Acad. Ophth. & Oto-laryngol., 1925.

35. A Demonstration of Ear, Nose and Throat Cases. M. Clin. North America (March), 1926.

36. Report of Special Committee on Tuning Forks and Tests. Tr. Amer. Acad. Ophth. and Oto-laryngol, 1926.

37. The Philosophy of the Older Tests of Hearing. Laryngoscope (June), 1927.

38. Recent Literature on Cranial Resonance and Its Clinical Application. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (June), 1927.

39. Studien über den Rinneschen Versuch mit besonderer Berücksichtigung der Anwendung des Stimmgabelgriffes sowohl bei Knochen- wie bei Luftleitung. Beitr. Anat., Phys., Path., Ther. des Ohres., Nase, Halses, v. 25, 1927.

40. Report of Committee on Tuning Forks and Tests of Hearing. Tr. Acad Ophth. & Oto-laryngol., 1927.

41. Signs and Symptoms of Common Nose, Throat and Ear Conditions, Including Defective Hearing. Elementary School J. (Feb.), 1928.

42. The Rationale of Tuning Fork Tests. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (March), 1928.

43. Report of Committee on Standardization of Tuning Forks and Hearing Tests. Tr. Amer. Acad. Ophth. & Otolaryngol., 1928.

44. Sound Meter. Tr. Am. Otol. Soc., 1928.

45. Presentation of a New Tuning Fork. Tr. Am. Otol. Soc., 1928.

46. The Use of Standardized Tuning Forks and Hearing Tests in the Military Service. Military Surg. (Feb.), 1929.

47. Some Very Accurate Measurements of the Amplitude of Vibration of Tuning Fork Prongs. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (March), 1929.

48. The Use of the New Magnesium Alloy Tuning Forks. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (Sept.), 1929.

49. Mixed Tumors in the Soft Palate. Arch. Otol. (Feb.), 1930.

50. The Essentials of Hearing Tests. J. Michigan Med. Soc. (May), 1930.

51. Einige neue Entwicklungen in Stimmgabeln und Tonstäben. Passow-Schaefer, Beiträge, v. 29, 1931.

52. The Indispensable Uses of Narcotics in the Practice of Otolaryngology. J. A. M. A. (April 18), 1931.

53. Problems of Pregressive Deafness of Adult Life, Including Etiology and Pathology of Otosclerosis. Auditory Outlook (Oct.), 1931.

54. Intrinsic Carcinoma of the Larynx, with a Consideration of Some Methods of Operative Approach. Illinois M. J. (Sept.), 1932.

55. Fundamental Principles of Functional Hearing Tests. Arch. Otol. (Nov.), 1933.

56. A Brief Consideration of the History of the Development of Mastoidectomy. S., G. & O. (Feb.), 1936.

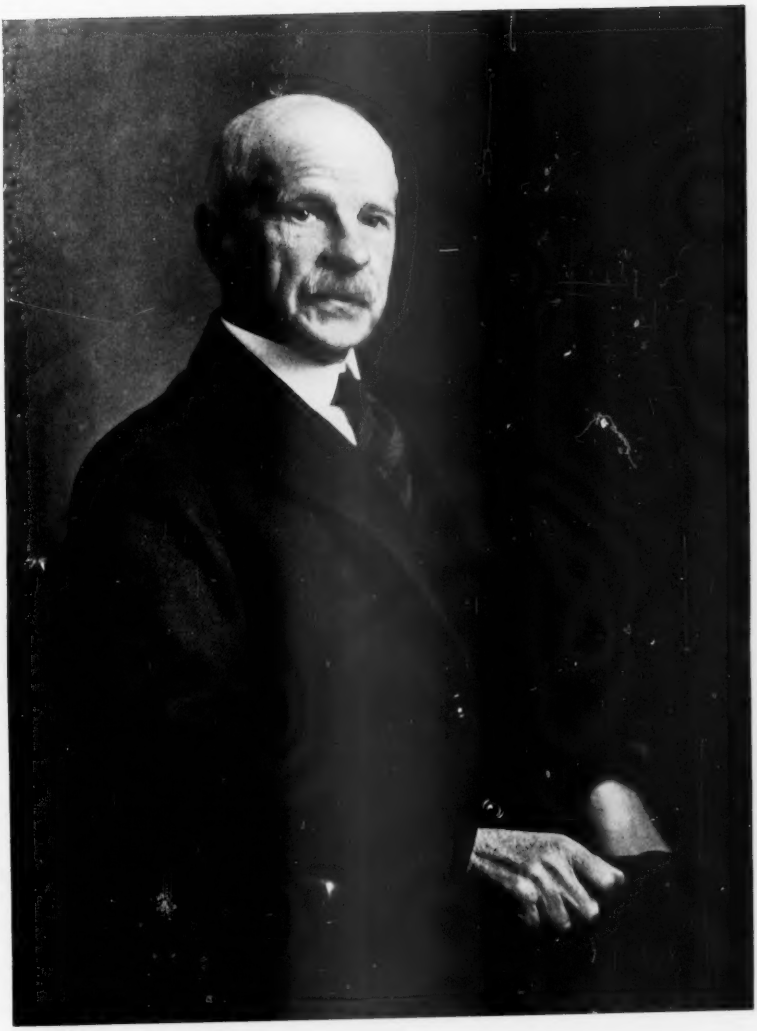
57. Practical Points in Hearing Tests and Selection of Hearing Aids. Illinois M. J. (Oct.), 1936.

58. A Brief Consideration of the History of the Development of Mastoidectomy. Ann. Med. Hist., 8:6, 1936.

59. Surgery of the Ear. Textbook of Surgery, by F. Christopher, 1936.

60. Hearing Tests. Cyclopedia of Medicine, 1936.

61-74. The Functional Examination of Hearing. Arch. Otol. Yearly Reviews, 1925-1938.



ARTHUR LOGAN TURNER

ARTHUR LOGAN TURNER

1865-1939

With the passing of Dr. Logan Turner in the summer of this year, Laryngology has lost another of its grand old men.

Known to Americans chiefly through his popular textbook "Diseases of the Nose, Throat and Ear," which first appeared in 1924 and has enjoyed several subsequent editions and printings, and his capable editorship of the Journal of Otology and Laryngology (1921-1930), he was the author of many important publications.

The present editors of the Journal credit him in large part with its development and establishment upon its present high plane. In a recent issue, his friend, Brown Kelly, writes feelingly of the man and his wide influence upon the medical thought of his day.

He was born in Edinburgh in 1865 and was the son of Sir William Turner, Demonstrator of Anatomy. Graduated in Medicine from the University of Edinburgh in 1889, he began his career as a general surgeon, but shortly transfered his attention to the diseases of the ear, nose and throat. He served as specialist to the Royal Infirmary from 1906, and at the expiration of his normal term was invited to continue as Honorary Surgeon Consultant.

In many of his endeavors he associated with him his distinguished assistant, the late J. S. Fraser, and in 1930, together with F. Esmond Reynolds, published a monograph "Intracranial Pyogenic Diseases," a collection of articles which had previously appeared in the Journal.

Many honors were bestowed upon him during his long life. On the occasions of his several visits to America, he was made an Honorary Member of the American Medical Association, an Honorary Fellow of the American Laryngological, Rhinological and Otological Society, and Corresponding Fellow of the American Laryngological Association.

His passing is a personal loss to his American colleagues.

The portrait of Dr. Turner is reproduced through the courtesy of the Journal of Otology and Laryngology.

Notices

Fourth International Oto-Rhino-Laryngological Congress

The Committee of the Fourth International Oto-rhino-laryngological Congress regret that they are obliged to give notice of their decision to postpone the Congress for an indefinite time.

Our first circular-letter was ready to be forwarded in August to the 8,000 Oto-Rhino-Laryngologists, whose addresses were known. It contains the information that H. M. the Queen of the Netherlands graciously had accepted the patronage of the Congress. Four Ministers, the Governor of the province of Noord-Holland and the Burgomaster of Amsterdam were to be Honorary Presidents; Delegates of H. M.'s Government were to hold a reception for the Congress in the Amsterdam National Gallery (Rijksmuseum) and the Netherlands O. R. Laryngological Society invited all members to the closing banquet.

We thankfully mention that for the three main subjects nine well-known colleagues had promised to deliver reports; besides over twenty Oto-Rhino-Laryngologists from several countries, had already announced papers.

We hope that the present horror will give way to better times and that at some future date it may be granted to us to send our invitation once more to the oto-rhino-laryngological world.

H. Burger, President.

A. Marres, Hon. Secretary.